A Dissertation on

# "LABORATORY RISK INDICATOR FOR NECROTIZING FASCIITIS (LRINEC) SCORE – AS A TOOL FOR DIFFERENTIATING NECROTIZING FASCIITIS FROM OTHER SOFT TISSUE INFECTIONS"

Dissertation Submitted to

**THE TAMIL NADU Dr.M.G.R. MEDICAL UNIVERSITY**

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with partial fulfillment of the regulations

for the award of the degree of

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# Coimbatore Medical College

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## ETHICS COMMITTEE

## CERTIFICATE

Name of the Candidate : DR. ALICE PRISCILLA

Course : POST GRADUATE IN M.S GENERAL SURGERY

Period of Study : ONE YEAR

College : COIMBATORE MEDICAL COLLEGE AND HOSPITAL

Dissertation Topic : LABORATORY RISK INDICATOR FOR NECROTISING FASCITIS [LRINEC] SCORE - AS A TOOL FOR DIFFERENTIATING NECROTISING FASCITIS FROM OTHER SOFT TISSUE INFECTIONS.

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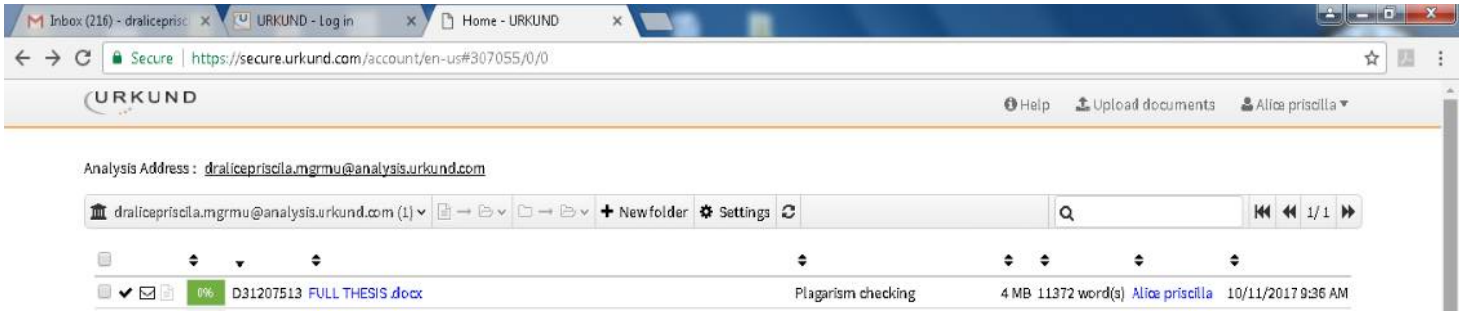
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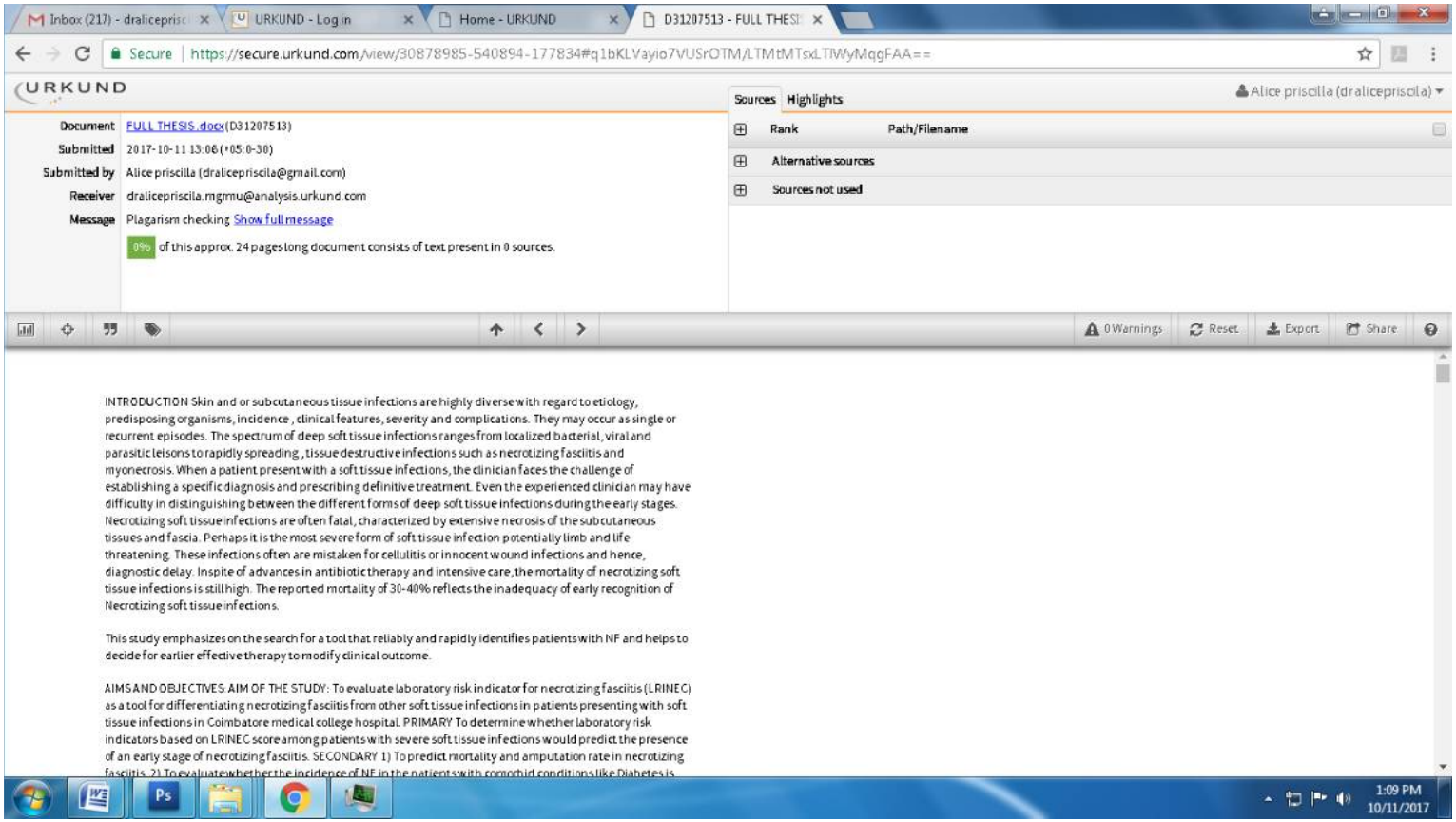
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INTRODUCTION Skin and or subcutaneous tissue infections are highly diverse with regard to etiology, predisposing organisms, incidence , clinical features, severity and complications. They may occur as single or recurrent episodes. The spectrum of deep soft tissue infections ranges from localized bacterial, viral and parasitic leisons to rapidly spreading , tissue destructive infections such as necrotizing fasciitis and myonecrosis. When a patient present with a soft tissue infections, the clinician faces the challenge of establishing a specific diagnosis and prescribing definitive treatment. Even the experienced clinician may have difficulty in distinguishing between the different forms of deep soft tissue infections during the early stages. Necrotizing soft tissue infections are often fatal, characterized by extensive necrosis of the subcutaneous tissues and fascia. Perhaps it is the most severe form of soft tissue infection potentially limb and life threatening. These infections often are mistaken for cellulitis or innocent wound infections and hence, diagnostic delay. Inspite of advances in antibiotic therapy and intensive care, the mortality of necrotizing soft tissue infections is still high. The reported mortality of 30-40% reflects the inadequacy of early recognition of Necrotizing soft tissue infections.
This study emphasizes on the search for a tool that reliably and rapidly identifies patients with NF and helps to decide for earlier effective therapy to modify clinical outcome.
AIMS AND OBJECTIVES AIM OF THE STUDY: To evaluate laboratory risk indicator for necrotizing fasciitis (LRINEC) as a tool for differentiating necrotizing fasciitis from other soft tissue infections in patients presenting with soft tissue infections in Coimbatore medical college hospital. PRIMARY To determine whether laboratory risk indicators based on LRINEC score among patients with severe soft tissue infections would predict the presence of an early stage of necrotizing fasciitis. SECONDARY 1) To predict mortality and amputation rate in necrotizing fasciitis. 2) To evaluate whether the incidence of NF in the patients with comorbid conditions like Diabetes is
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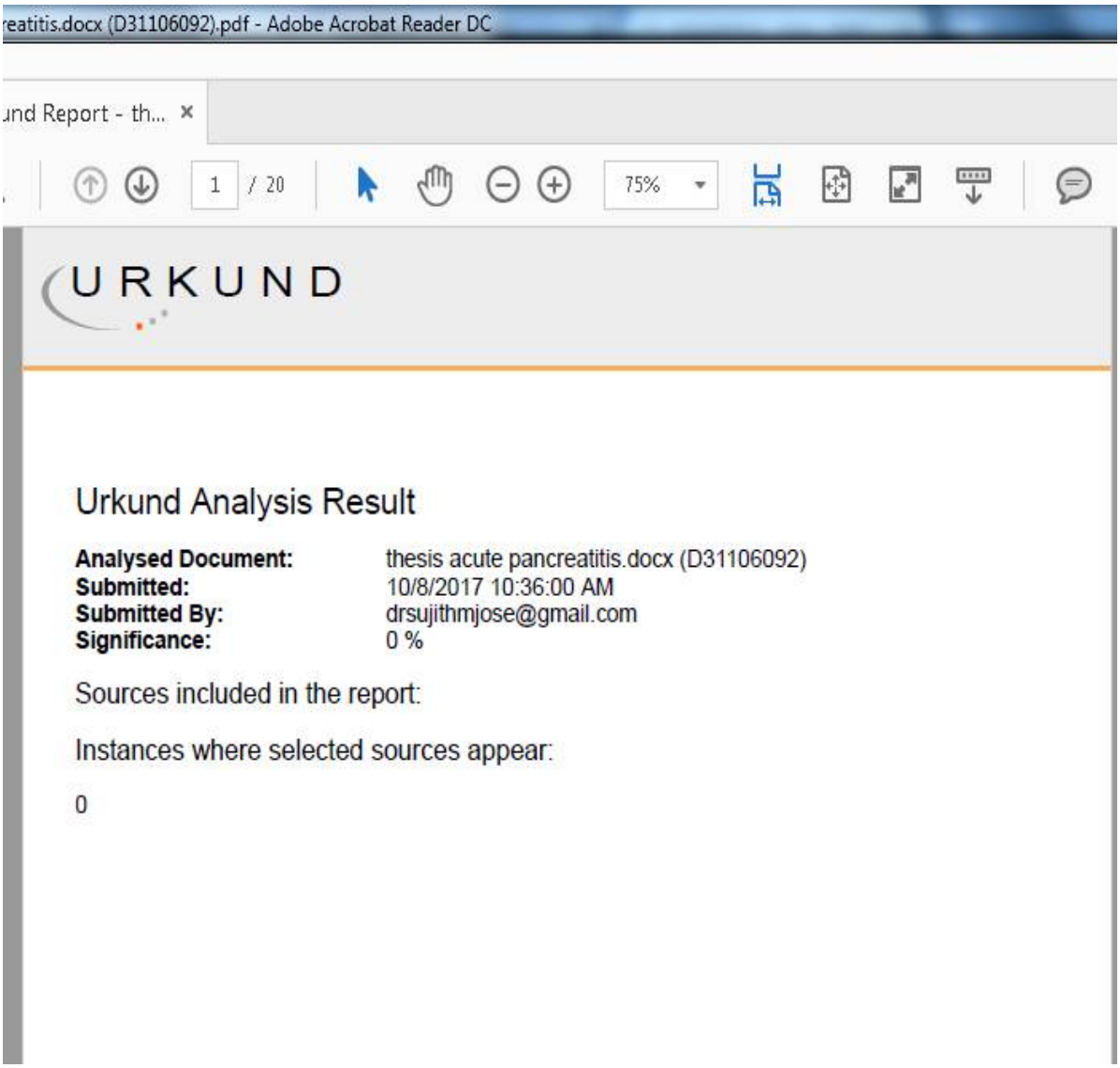
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## CERTIFICATE

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**HOD**
**Prof. Dr. V. Elango M.S.,**
Head of the Department
Department of General Surgery
Coimbatore Medical College
Coimbatore

**GUIDE**
**Prof.Dr.K. Santhi MS.,**
Professor of Surgery
Department of General Surgery
Coimbatore Medical College
Coimbatore

**Dr.B.Asokan M.S., MCh.,**
The Dean
Coimbatore Medical College and Hospital
Coimbatore

## DECLARATION

The dissertation titled **"LABORATORY RISK INDICATOR FOR NECROTIZING FASCIITIS (LRINEC) SCORE – AS A TOOL FOR DIFFERENTIATING NECROTIZING FASCIITIS FROM OTHER SOFT TISSUE INFECTIONS"** is being submitted by me to "The Tamil Nadu Dr. M.G.R. Medical University" in partial fulfillment of the regulation for the completion of the M.S General Surgery Degree Examination to be held in 2018. This work has been carried out in the Department of General Surgery, Coimbatore Medical College and Hospital, Coimbatore under the guidance of Dr.K. Santhi M.S., Professor of General Surgery, Coimbatore Medical College and Hospital, Coimbatore.

Date:
Place: Coimbatore Dr. Alice Priscilla

## CERTIFICATE – II

This is to certify that this dissertation work titled **“LABORATORY RISK INDICATOR FOR NECROTIZING FASCIITIS (LRINEC) SCORE – AS A TOOL FOR DIFFERENTIATING NECROTIZING FASCIITIS FROM OTHER SOFT TISSUE INFECTIONS"** of the candidate **DR. ALICE PRISCILLA** with registration Number **221511301** for the award of **M.S in the branch of General Surgery**, I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains 96 pages from introduction to conclusion and the result shows **0% (Zero)** percentage of plagiarism in the dissertation.

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**Dr. Alice Priscilla**

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# INTRODUCTION

Skin and or subcutaneous tissue infections are highly diverse with regard to etiology, predisposing organisms, incidence , clinical features, severity and complications.

They may occur as single or recurrent episodes. The spectrum of deep soft tissue infections ranges from localized bacterial, viral and parasitic leisons to rapidly spreading , tissue destructive infections such as necrotizing fasciitis and myonecrosis.

When a patient present with a soft tissue infections, the clinician faces the challenge of establishing a specific diagnosis and prescribing definitive treatment. Even the experienced clinician may have difficulty in distinguishing between the different forms of deep soft tissue infections during the early stages.

Necrotizing soft tissue infections are often fatal, characterized by extensive necrosis of the subcutaneous tissues and fascia. Perhaps it is the most severe form of soft tissue infection potentially limb and life threatening. These infections often are mistaken for cellulitis or innocent wound infections and hence, diagnostic delay. Inspite of advances in antibiotic therapy and intensive care, the mortality of necrotizing soft tissue infections is still high. The reported mortality of 30-40% reflects the inadequacy of early recognition of Necrotizing soft tissue infections.

This study emphasizes on the search for a tool that reliably and rapidly identifies patients with NF and helps to decide for earlier effective therapy to modify clinical outcome.

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### AIM OF THE STUDY:

To evaluate laboratory risk indicator for necrotizing fasciitis (LRINEC) as a tool for differentiating necrotizing fasciitis from other soft tissue infections in patients presenting with soft tissue infections in Coimbatore medical college hospital.

### PRIMARY

To determine whether laboratory risk indicators based on LRINEC score among patients with severe soft tissue infections would predict the presence of an early stage of necrotizing fasciitis.

### SECONDARY

1) To predict mortality and amputation rate in necrotizing fasciitis.

2) To evaluate whether the incidence of NF in the patients with comorbid conditions like Diabetes is increasing or not.

## REVIEW OF LITERATURE

## ANATOMY

### SKIN:

Skin is occupying the larger surface area of the body than all the organs and consists of 16% of weight of the body. It has two layers, the outer layer epidermis and dermis the inner layer .

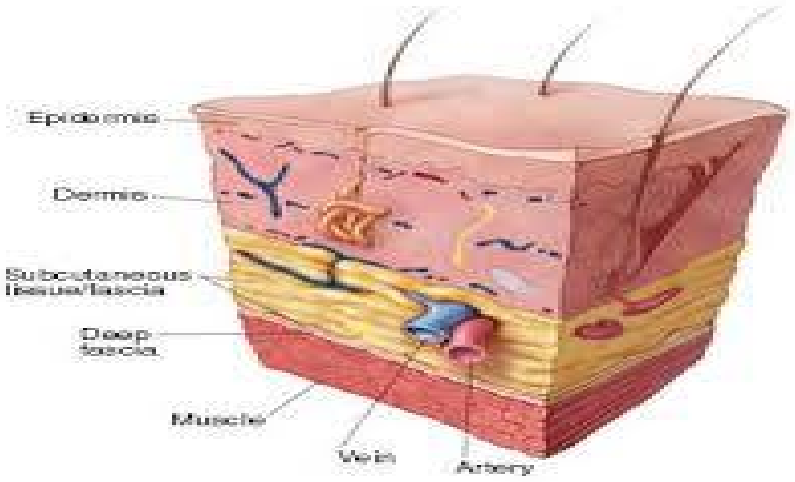



**Fig 1.anatomy of skin and subcutaneous tissue**

**There are five layers of cells that are present in the epidermis:**

1-stratum corneum layer (keratin layer)

2-stratum lucidum present in soles and palms only

3-stratum granulosum ( granular cell layer)

4-stratum spinosum (prickle cell layer)

5-stratum basale (keratin layer)

Immediately beneath the epidermis is the dermis. It is a richly vascular layer which has many receptors which are sensory. This layers prevents the entry of certain substances like chemicals and also gives support mechanically to the overlying epidermis.

**COMPOSITION:**

1-Connective tissue elements like (collagen/elastin)

2- Epithelium containing skin appendages which are secondary like sweat glands and follicles of hair.

3-Nerves which includes sensory Nervous structures like Pacinian corpuscles and Meissner corpuscles. The external surface of the skin made up of squamous epithelial cells which are keratinised. The picture shown below shows the various skin and soft tissue infections involving the various anatomical layers of skin and subcutaneous tissue.

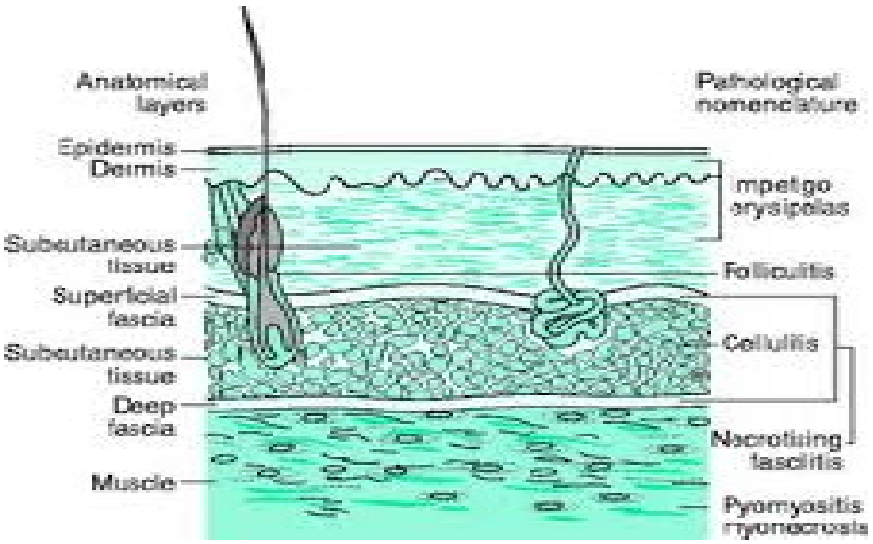



**Fig 2. pathological nomenclature involving various anatomical layers**

## SUBCUTANEOUS TISSUE

The subcutaneous tissue otherwise known as hypodermis subcutis, hypoderm or superficial fascia is the most lower layer of the integumentary system in humans.

The cells that are found in the hypoderm are fibroblasts, adipose cells and macrophages. The hypoderm is the derivative of the mesoderm. Its usage is storage of fat lies beneath dermis which lies beneath epidermis. It comprises mainly loose connective tissue & fat lobules.

It is having blood vessels which are larger and nerves than those found in the dermis.

**COMPOSITION:**

- Body which are fibrous in nature connecting the skin to deep fascia.
- Collagen and elastin fibres which attaches the tissue to the dermis.
- Fat except in the penis, pinna, eyelids, clitoris.
- Blood vessels that track from dermis.
- Lymphatic vessels that track from dermis.
- Part of some sweat gland, that is Mammary gland also prevent completely with in this tissue, they are also called as Modified apocrine sweat glands.
- Roots of the hair follicles.
- Skin nerve and free nerve endings.

- Bursa is present in the tissue overlying joints to provide smooth passage of the above present skin.
- Mast cells
- Ruffini & Pacinian corpuscles.

**SUBCUTANEOUS FAT:**

The most largely distributed tissue layer which is subcutaneous is subcutaneous fat. It is composed of group of adipocytes in lobules separated by fibrous connective tissue. The count of adipocytes may vary among various area of the body and the size also varies according to the nutritional state of individual body.

Its functions as padding and provides energy reserve and thermoregulation.

**EPIDEMIOLOGY**

Necrotizing fasciitis is an acute rapidly spreading infection of subcutaneous soft tissue involving both the superficial and deep fascia. It causes deadly events in the affected individual.

In BC 5th century, Hippocrates first mentioned that erysipelas was turned to a complication of Necrotizing fasciitis. Necrotizing fasciitis has been mentioned in medical and surgical text since 18th century.

In 1920, 20% of patients were reported by Meleney and afterwards, these leisons were given the term Meleney’s gangrene. In

1952 the term Necrotizing fasciitis was coined by Wilson.

As the incidence of malignancy, diabetes and other diseases which causes immunosuppression increases, the incidence of Necrotizing fasciitis also increases. But the cases which are presented to us is the tip of iceberg .These soft tissue infections can affect all the age groups like in children it can occur as impetigo and in adults it can occur erysipelas.

During the war seasons till the middle of 20th century, injuries were infected by Clostridium spp. which causes gas gangrene. In USA during the civil war almost 50% of the soldiers who got gunshot injuries developed soft tissue infections predominantly gas gangrene. Factors contributing to that are severe trauma, inadequate surgical debridement, dirty or untidy wounds, improper dressings, etc..

The most common organisms found on these infections at that time was Group A streptococci , Clostridium perfringens, Gram negative bacteria and mixed organisms.

In the past time of first world war in Europe, where there was the presence of Clostridial spores in the soil enriched with animal feces, the injured individuals developed gas gangrene .But gas gangrene cases were found to be reported less in North Africa where the sand in the deserts had few clostridial spores.

But during our modern times, gas gangrene cases are decreasing because of immediate first aid , surgical intervention in well equipped hospitals, development of broad spectrum antibiotics.

In the recent times, in intravenous and intradermal drug users, Cl.sordeii, Cl.perfringens and Cl.novyi are found to be causing soft tissue infections.

Necrotizing fasciitis is a deadly soft tissue infection which can present alone or in combination with gas gangrene. The other superficial infection of the skin are cellulitis, impetigo, erysipelas, carbunculosis and furunculosis

**PATHOGENESIS**

Our skin is exposed to various stimuli both externally and internally, infections, chemical agents and skin produces a various response by various means. The capillary plexus underneath the papillae of dermis gives nutrition to the dermal cells and the dermatocytes that are tightly bound together develops protective layer from invading microbes.

Once microbes entered through the trauma , cut injury, under the hair follicle ,there will be the inflammatory response of the host in the infection site by delivering oxygen, complements, antibodies, macrophages, etc.

In addition to the above, there will be the release of proinflammatory cytokines which in turn causes fever, rise in the antibody levels and rise in the production of C-reactive protein like acute phase reactants.

The cytokines stimulate endothelial cells and these cells release nitric oxide and prostaglandins which in turn produces vasodilatation thereby causing increased flow of blood to the injured site.

These changes produces the signs of inflammation

1.Erythema
2.Swelling
3.Heat
4.Tenderness or pain

**Fig.3 Signs of Inflammation**

If there is a moderately increased tissue perfusion, tissues may remain viable, but there will be lowering of threshold for the progression of infection.

Predisposing conditions are

1. Peripheral vascular disease involving large arteries

2. Diabetes mellitus producing microvascular disease

3. Chronic venous stasis causing post capillary obstruction

Though most bacteria and fungi multiply in the tissue which are viable, the fibrous attachments in between subcutaneous tissues and fascia limits the spread of infection. The naturally lacking fibrous attachments leads to fast spread of infection in the extremities or in the trunk. The primary site of pathology in this condition is the superficial fascia. The proposed mechanism of spread is due to production of hylauronidase like enzymes from bacteria which causes the degradation of fascia. The excessive multiplication of bacteria will produce angiothrombotic invasion of microbes and liquefactive necrosis of the fascial tissues which lies superficially.The spreading infection along the fascial planes leads to nutrient vessels thrombosis to skin and the skin ischemia and necrosis

In this pathogenesis first there is a radial phase in which the spread of infection along the fascia predominates and then followed by undermining of normal skin

If this condition progresses ,there is a development of ischemic necrosis of skin and gangrene of skin and subcutaneous tissue along with

bullae formation, dermal necrosis and ulceration. Skin and deeper tissue necrosis will develop due to severe hypoxia. Examples are

1. Decubitus ulcers due to pressure necrosis.

2. Compartment syndromes resulting in hypoxia and muscle necrosis within tight bundles of fascia.

**Fig.4 Skin Necrosis with Ulceration**

## CLASSIFICATION OF DEEP SOFT TISSUE INFECTIONS

### CLINICAL TYPES ARE TWO

### Type 1

Type 1 is polymicrobial mainly due to a mixed aerobic bacteria and anaerobic bacterial infection. It is commonly seen in diabetic patients, postsurgical patients and in patients with peripheral vascular disease.

### Type 2

Type 2 is monomicrobial due to group A streptococci and called as streptococcal gangrene. It can occur in any age group in contrast to type 1. Of recent years there has been a dramatic increase in the number of

invasive infections caused by group A streptococci including necrotizing fasciitis. Moreover Patients who do not have complicated medical illness may develop type 2 necrotizing fasciitis

**PREDISPOSING FACTORS**

- Intravenous drug abuse
- History of blunt trauma
- Nonsteroidal antiinflammatory agents
- Childbirth
- Chickenpox
- Muscle strain
- Wounds such as caused by laceration or a surgical procedure

Classification based on etiology

- Primary or idiopathic
- Secondary

If the cause is unknown or unidentifiable etiologic factor, it is known as primary or idiopathic NF. In view of early diagnosis and management, it is wiser to consider that idiopathic NF exists and it is a distinct clinical entity.

If the etiology is known, these are called as secondary NF. The entry of bacteria occurs following predisposing events such as cuts, abrasions, contusions, lacerations, burns, subcutaneous injections, bites, or operative

incisions that causes break in the epidermis. In circumstances of occult infection such as infected Bartholin's cyst , perforated hollow viscus or as a complication of perirectal abscess, secondary NF can also occur.

**CLINICAL FINDINGS**

Necrotizing fasciitis is a severe infection of the subcutaneous soft tissues principally the superficial and often the deep fascia. Generally it is an acute eventual process but rarely it may present as a subacute progressive disease.

Any part of the body can be affected by Necrotizing fasciitis but most commonly in the long extremities. The other areas of predilection are the perianal and groin areas, abdominal wall and postoperative wounds. The gateway of infection is from trauma sites such as laceration, abrasion ,insect bite, burns, a laparotomy performed in the presence of peritoneal soiling (penetrating abdominal trauma or perforated viscus) or another surgical procedure (eg.vasectomy), perirectal abscess, decubitus ulcer or intestinal perforation .The bowel perforation may be due to diverticulitis, carcinoma of the rectosigmoid junction or a foreign body such as toothpick or chicken pox. Necrotizing fasciitis from the intestinal sources may occur in the groin, abdominal wall or in the lower extremity. The spread of infection from the intestinal sources to the lower extremity is via extension along the psoas major and to that of abdominal wall is via a colocutaneous fistula. In particular necrotizing fasciitis develop in the

clinical setting of alcoholism , diabetes mellitus and parenteral drug abuse.

The region involved is initially swollen, reddened, shiny with irregular margins, hot, extremely tender and painful . Lymphangitis and lymphadenitis are uncommon.

The disease process rapidly progresses over many days ,with the sequence of integument colour changes from patches of purple red to bluegrey. In 3 to 5 days after the onset of infection, the skin breaks down with the formation of bullae (containing thick pink or purple coloured fluid and frank cutaneous gangrene(resembling a thermal burn) can be seen.

After this time the involved area is no longer tender and becomes anaesthetic due to microvascular thrombosis and superficial nerves destruction that are usually present in the necrotic, undermined subcutaneous tissue. In times the onset of loss of sensation in the involved area comes before the appearance of skin necrosis. This suggests that the process involved is necrotizing fasciitis and not a simple resolving cellulitis.

Marked edema and swelling may lead to compartment syndrome with extensive muscle necrosis which may require prompt fasciotomy. Measurement of pressure inside the compartment may aid the evaluation

in early situations in which marked pain and the swelling are present without concomitant skin changes that would indicate the diagnosis.

The gas in the subcutaneous tissue is frequently present in the necrotizing fasciitis with polymicrobial form seen commonly in patients having diabetes mellitus. In this systemic toxicity is prominent along with the elevation of temperature in the range of 38.9 C to 40.5C. On probing of the leison with a haemostat through a small incision, the instrument passes easily along a plane just above the deep fascia. Such easy passage would not occur with simple cellulitis.

The Centre for Disease Control and Prevention and The National Necrotizing Fasciitis Foundation has compiled the following as symptoms of NF :

**Early symptoms of NF (within 24 hours):**

- Commonly a trivial trauma or breech of skin has occurred(not necessarily appear infected)
- Pain in the injured site .Pain may sometimes be in the same region or limb of the body
- Pain often disproportionate to the grade of injury
- Flu like illness then faces up with symptoms such as nausea, diarrhoea, dizziness, weakness, fever, confusion and malaise
- Dehydration

- The biggest symptom is combined of all these symptoms

**Advanced symptoms(usually within 3 to 4 days)**

- The injured area swells and the development of purplish may occur
- Development of blisters with blackish fluid
- The wound may appear necrotic, associated with a dark mottled flaky appearance.

**Critical symptoms (usually within 4 to 5days)**

- Hypotension
- State of septic shock from the toxins released by the bacteria
- Unconsciousness

**CLINICAL DIAGNOSIS**

Early recognition of disease characteristic features and the rapidly progressive clinical course of the disease aids in the diagnosis of necrotizing fasciitis

**Table.1 Clinical stages of necrotizing fasciitis**

| Stage1 | Stage2 | Stage3 |
|---|---|---|
| Tenderness | Blisters and bullae formation | Tissue necrosis |
| Erythema | | Hyposensitivity |
| Oedema | | Anaesthesia |
| Warm skin | | Tissue crepitation |
| Fever | | Haemorrhagic bullae |

Preceding history of soft tissue injury could be there commonly from a penetrating or blunt trauma, animal or insect bite, postoperative infection, minor skin infection or even injections that the patient has received as in subcutaneous insulin or illicit drugs

The diagnostic characteristics which are overlapping between cellulitis and necrotizing fasciitis initially may mislead the diagnosis of NF to cellulitis .This results in the delayed management of such sever condition underneath.

Not uncommonly pain out of the proportion to the elicited sign is the only early differentiating feature. The infection begins at the junction of dermis and superficial fascia , rather than in case of NF, it starts at the level of subcutaneous fat and deep fascia.

In the early stages of NF , sparing of the epidermal and dermal layers is present. There is no obvious erythema and edema of the epidermal and dermal layers initially. If subcutaneous fat necrosis is extensive, hypocalcemia may occur.

A number of symptoms and signs that have been proposed may help in differentiating the two above mentioned conditions. A Canadian study outlined patients with necrotizing fasciitis as more likely to have a generalised erythematous rash and a toxic apppearence. The cytolysins and the toxins which are pyrogenic are responsible for hypotension,

disseminated intravascular coagulation and multi organ failure.

Radiological studies help in assessing the extent of infection in the tissues, the presence of gas In the subcutaneous tissues helps in determining the infection extent, especially in mixed aerobic and anaerobic or clostridial infections. Plain radiography may help in the detection of gas in the soft tissues. But Magnetic Resonance Imaging and Computer Tomography are more superior in revealing the extent of infection


**Fig.4. Plain radiograph showing subcutaneous gas in necrotizing fasciitis**

The features of necrotizing fasciitis in CT scan include enhancement and thickening of deep fascia, the presence of fluid and gas in the soft tissue planes in and around the superficial fascia.

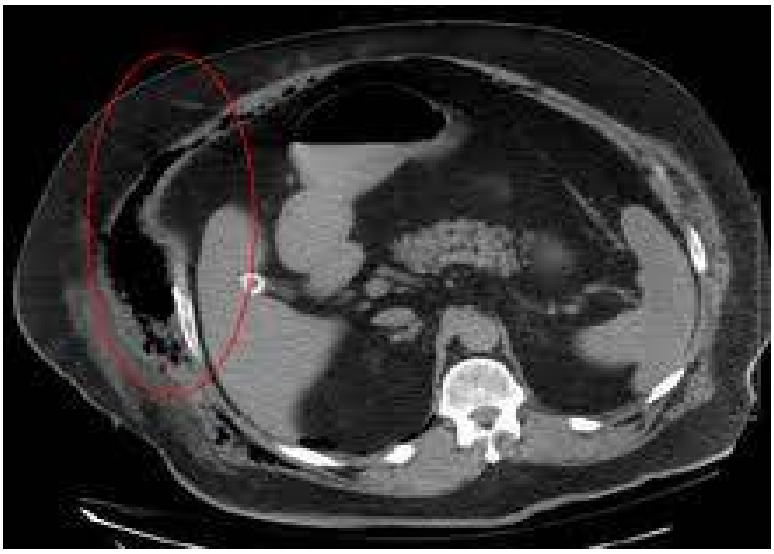


**Fig. 6 CT showing gas in the subcutaneous tissue in necrotizing fasciitis**

In USG the features of NF are distortion and thickening of the deep fascia and collection of fluids along the deep fascia.

CT is inferior to MRI in differentiating necrotic and necrotic tissue .In MRI the features that are distinct for NF includes fluid collections in the deep fascia and thickening and hyper intense T2W signal within the muscles.

In MRI ,the sensitivity often exceeds the specificity that ensues in overestimation of extent of deep fascial involvement on MRI almost certainly excludes NF.

However the cost and availability of this limits the routine use of Computed tomography, Magnetic Resonance Imaging and frozen section biopsy in the evaluation of NF. Hence, Wong et al designed a simple

scoring system, the laboratory risk indicator for necrotizing fasciitis (LRINEC) which is based on more readily available investigations at most centers, and that helps in distinguishing necrotizing fasciitis from other soft tissue infections.

**Fig.7.MRI showing the presence of subcutaneous gas in necrotizing fasciitis affecting left lower limb**

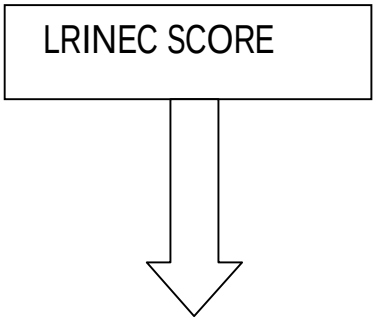
The LRINEC score is calculated based on points assigned for six laboratory variables at the time of presentation including; C-reactive protein , blood hemoglobin, total leucocyte count, serum glucose, serum sodium, serum creatinine.

**Table 2. Laboratory Risk indicator for Necrotizing fasciitis (LRINEC ) score**

| Variable | Score |
|---|---|
| C reactive protein | |
| Pos ≥ 150mg/dl | 4 |
| Neg <150 mg/dl | 0 |
| Total White cell count x $10^3$ permm$^3$ | |
| <15 | 0 |
| 15 -25 | 1 |
| >25 | 2 |
| Hemoglobin, gm/dl | |
| >13.5 | 0 |
| 11 -13.5 | 1 |
| <11 | 2 |
| Sodium, mmol/l | |
| ≥ 135 | 0 |
| <135 | 2 |
| Creatinine, mg/dl | |
| ≤ 1.6 | 0 |
| >1.6 | 2 |
| Glucose, mg/dl | |
| ≤ 180 | 0 |
| >180 | 1 |

The maximum score is 13

The LRINEC score stratifies patients with soft tissue infections into three categories, low, intermediate and high risk for necrotizing fasciitis even if there is a equivocal clinical picture.




1. LOW RISK LESS THAN OR EQUAL TO 6
2. INTERMEDIATE RISK 7,8
3. HIGH RISK MORE THAN 8

| LRINEC Score | Inference |
|---|---|
| ≤6 | Low risk |
| >6 | Suspicious of NF/intermediate |
| >8 | Strong prediction of NF/high risk |

Although Wong et al suggested LRINEC score as capable of detecting even clinically early cases of NF, other studies did not yielded satisfactory results so far to validate LRINEC score for routine score.

**SURGICAL DIAGNOSIS**

Tissue biopsy collected during wound exploration and surgical debridement remains the gold standard in the diagnosis of necrotizing

fasciitis. In NF, tissue diagnosis is made only if the fascia is infiltrated by polymorpho nuclear leukocytes.

Tissue intergrity and depth of invasion while doing wound exploration can also be evaluated. The presence of myonecrosis and fascial necrosis indicates the presence of necrotizing infection. Definitive features of loss of fascial intergrity along the fascial planes and the involvement of muscles are also diagnostic

A bedside procedure that helps in diagnosis is the finger test. Under local anesthesia, a 2 cm incision is made down to the deep fascia and a gloved finger is then probed at the level if the superficial fascia. Foul smelling dishwater pus, lack of bleeding and least tissue resistance to finger dissection signifies a positive finger test and are considered to be diagnostic of necrotizing fasciitis.

**MICROBIOLOGY**

The exudate collected from the wound on gram staining usually shows a mixture of organisms or chains of gram-positive cocci in case of streptococcal gangrene,.

Infection with a single pathogen occurs in only 15 % to 29% of patients. NSTIs are typically polymicrobial in nature.

Staphylococcal and streptococcal species are relatively common causative organism in combination with anaerobes. Vibrio and fungal

pathogens have also been described as causing pathogen.

Clostridial infections are typically monomicrobial, although they can be seen in combination with other bacteria. They are usually characterized by the presence of necrosis in the muscles. And the patients having these type of infections are having poor prognosis. The organisms commonly with Clostridial infections are Clostridium perfringens, C,septicum and C. novyi. In most of the studies, along with the several members of Enterobacteriaceace , Clostridium difficle has also been isolated from the infected wound drainage material.

Commonly cultured gram positive organisms are coagulase negative staphylococci, staphylococcus aureus, group A haemolytic streptococci, sta. epidermidis, enterococccci and clostridial species.in 10% of the patients mixed gram positive organisms are present on the infection. The common gram negative organisms associated with these infections are Enterobacter species, Escolar, proteus sp, Pseudomonas, bacteroides, Serratia sp and mixed gram negative organisms .The only bacteria commonly reported as the sole cause of non clostridial NSTIs is **β**-haemolytic streptococcus pyogenes, so called flesh eating bacteria and thus it causes a rapidly spreading NSTIs.

## COMPLICATIONS

1. Delay in diagnosis
2. Bacteremia
3. Delay in first debridement
4. Acute renal failure
5. ARDS/ other respiratory failure
6. Inadequate debridement
7. Multi organ system dysfunction
8. Other infection (eg. pneumonia, UTI )
9. Clostridium difficle colitis
10. Decubitus ulcer/ break down
11. Seizure
12. Iatrogenic source of necrotizing fasciitis
13. Antibiotic reaction
14. Osteomyelitis
15. Cardiopulmonary arrest
16. Feceal wound spillage
17. Heart failure
18. Complications from HBO therapy

Acute renal failure is defined as a rise in serum creatinine to more than twice the base line level or at least greater than 2 mg/dl.

Adult respiratory distress syndrome is defined as radiographic evidence of diffuse pulmonary edema not found to be cardiogenic, and is associated with a PaO2/FiO2 ratio of less than 150 and decreased compliance

Multiorgan system dysfunction defined as objective evidence of acutely diminished function in two or more organ systems that requires urgent medical or surgical intervention.

Complications from HBO therapy were uncommon which includes hypoglycemia, seizures, haemotympanum and claustrophobia.

**PREVENTION**

Centre for Disease Control has reported the following list of recommendations to prevent the disease.

- Patients with streptococcal throat infections should stay home until 24 hours after their antibiotic dose.
- Patients with sore throats should consult a doctor
- Proper hand wash do prevent the spread of Group A streptococcal infection especially before preparing food or eating ,after sneezing and coughing
- Keeping the skin intact is essential
- Patients with infected wounds and fever should seek early medical care
- Wounds should be cleaned and monitored regularly for signs of infection (redness, swelling, discharge, pain)

**MANAGEMENT**

The three essential steps in treatment are surgical debridement, intensive supportive care and appropriate antibiotics. Some patients

require mechanical ventilation and other patients require hemodialysis. Gross fall in BP an diffuse capillary leak often necessitates enormous amount of intravenous fluids (10 to 20 liters / day),although anasarca is a common complication .In some patients blood pressure improves with intravenous fluid alone .Sometimes vasopressors such as dopamine may be useful, but there is meager information from clinical studies in this specific infection. Although potent vasoconstrictors lie epinephrine may improve blood pressure, symmetric gangrene may ensue, partly as a result of the drug and partly as a result of poor perfusion caused by the bacteria, toxins and endogenous mediators.

The Surviving Sepsis Campaign, a multidisciplinary approach that worked to develop treatment recommendations has published guidelines incorporating evidence –based treatment strategies most recently in 2008. These guidelines are summarized as below.

Initial evaluation of infection issues.

**Initial resuscitation:**

Resuscitation should be started immediately in patients having hypotension or elevated serum lactate with resuscitation goal of CVP 8-12 mmHg, mean arterial pressure of ≥65mmHg and urine output of ≥0.5 ml/kg per hour.

**Diagnosis**

Obtain samples for cultures before antibiotic but antibiotic therapy not be delayed

**Antibiotic therapy**

Begin IV antibiotic therapy as early as possible should be within the first hour after recognition of severe sepsis/ severe shock. Use broad spectrum antibiotic regimen with penetration into presumed source. Reassess regimen daily. Discontinue antibiotics in 7 to 10 days for most infections, stop antibiotics for noninfectious issues.

**Source control**:

The anatomic site of infection should be established earlier. After initial resuscitation implement source control measures as soon as possible. Potentially infected intravascular access should be removed.

**HEMODYNAMIC SUPPORT AND ADJUNCTIVE THERAPY**

**Fluid Therapy**

Resuscitate using crystalloid or colloid using fluid volumes of 1000 ml (crystalloid), target CVP of 8-12 mmHg

**Vasopressors/ inotropic therapy**

Maintain MAP of ≥65 mmHg, centrally administered nor epinephrine or dopamine are first line choices, dopamine should not be used for renal protection, insert arterial catheters for patients requiring vasopressors, Do not increase he cardiac index to predetermined supra normal levels.

**Steroids**

Consider IV hydrocortisone (adult dose <=300 mg/d) for septic shock when hypotension is refractory to fluids or vasopressors.

**CVP –central venous pressure**
**MAP -mean arterial pressure**
**PEEP –positive end expiratory pressure**
**rhAPC – recombinant human activated protein C**

**Recombinant human activated protein C :**

Consider rhAPC in adult patients with sepsis-induced organ dysfunction. and high risk of death.

**OTHER SUPPORTIVE THERAPY**

**Administration of blood product;**

Packed cell transfusion if hemoglobin decreases to < 7 gm/dl

Mechanical ventilation Target an initial tidal volume of 6 ml/kg body weight and plateau pressure of <=30 cm H2O in patients with acute lung injury .Use PEEP to avoid lung collapse. Use a weaning protocol to evaluate the potential for discontinuing mechanical ventilation, Pulmonary artery catheter is not indicated for routine monitoring

**Glucose control**

Control hyperglycemia in patients with sepsis with IV Insulin

**Prophylaxis**

Use proton pump inhibitor or H2 blocker for stress ulcers and low dose unfractionated or fractional heparin for deep venous thrombosis prophylaxis

**Limitation of support**

Patients and families should be aware of advance care planning

**Nutritional support**

Aggressive nutritional support is compulsory in all patients after surgical debridement

Recommended allowances for calories would be double their basic calorie requirements given parenterally or orally. Nutritional support runs the risk of less complications with decreased mortality and morbidity rates,

**Pain control**

Per and postoperative pain control becomes the important part of management and it should be individualized for each patient

**PHARMACOTHERAPY**

Selection of antibiotics becomes more difficult in patients who are having the rapidly progressive infection. Antibiotic therapy of NSTIs is specifically directed to give broad spectrum coverage for gram positive, gram negative organisms and anaerobes.

The choice of empirical antibiotics is controversial and is dependent primarily on personal preference. In many patients ,the most commonly use regimens are(1) penicillin and an aminoglycoside plus clindamycin

(2) imipenem – cilastatin

(3)ampicillin plus sulbactam plus an aminoglycoside

Some practitioners select an antipseudomonal penicillin or vancomycin for penicillin allergic patients. Initially started empirical treatment can be adjusted according to bacterial sensitivities on tissue cultures.

Recent studies describes that clindamycin is better to penicillin for treatment of experimental necrotizing fasciitis and myonecrosis caused by group A Streptococci. It seems that penicillin failure is due to the decreased expression of penicillin binding proteins during the stationary phase of bacterial growth.

Clindamycin may be more efficacious because.

- It suppresses the production of toxin
- Its post antibiotic effect is long
- It is not affected by inoculum size or stage of growth
- M-protein synthesis is inhibited and phagocytosis of streptococcus pyogenes is facilitated.

Neutralization of toxins is a desirable therapeutic goal and is advocated by some experts. Some authorities recommend also giving immunoglobulin 400 mg/kg/d intravenously for 5 days for documented streptococcal toxic shock syndrome.

**SURGICAL INTERVENTION**

The major emphasis is inevitably surgical .Suspicion should be directed toward any wound incurred out of doors and contaminated with a foreign body, soil, f feces any wound in which tissue (particularly muscle) has been extensively injured. This type of wound should be carefully examined with the patient under sufficient anesthesia to permit full inspection and debridement of devitalized tissue , including muscle.

It is often difficult to distinguish necrotic from edematous tissue. Careful daily inspections of the wound will determine whether repeated debridement will be necessary. Daily debridement under anesthesia may be required, since these leisons are extensive and the degree of tissue viability is often difficult to assess in the operating room. tight fascial compartments must be compressed. Wide open drainage is essential and may require extensive denudation.

A functional extremity can be salvage usually in necrotizing fasciitis, if not amputation of the affected portion of the extremity can be safely performed later.

**FIG 8. Raw area left leg after surgical debridement**

It is important to avoid confusing fasciitis with deep gangrene. It is a tragic error to amputate an extremity when removal of dead skin and fascia will suffice. Immediate amputation is necessary when there is diffuse myositis with complete loss of blood supply or when adequate debridement would clearly leave an useless limb.

When the viability of the remaining tissue is confirmed and the infection has been controlled with sensitive antibiotics, raw area in the soft tissue can be covered with skin grafts.

**Fig 9. . Skin graft applied to cover the soft tissue deficit**

**The following factors favours limb salvage surgery**

**Table. 3 Factors favoring limb salvage surgery versus amputation**

| Limb salvage surgery | Amputation |
|---|---|
| Good past health | Concurrent medical disease with high anesthetic risk from multiple operations (eg .poorly controlled diabetes,valvular heart disease) |
| Not life threatening state | Myonecrosis |
| Multiple sites | Unremitting shock |
| Response to inotropic support | Concurrent peripheral vascular disease<br>Rapidly progressive infection<br>Large area of tissue necrosis (heel pad) |

## HYPERBARIC OXYGEN THERAPY

In the management of Clostridial myonecrosis or gas gangrene ,hyperbaric oxygen therapy is generally regarded as an important adjunct that can be used. The principle of HBO is that all necrotizing infections have decreased oxygen tension in the tissues, ischemia, and subsequent reduction in the host cellular immunity. Increased oxygen partial pressure has been associated with the reversal of basic pathophysiologic mechanism of necrosis. Increased oxygen tension reverses ischemia and thereby host defense mechanisms is also improved. Furthermore the transfer of drugs across the bacterial cell wall is also facilitated and hence enhances the action of various antibiotics

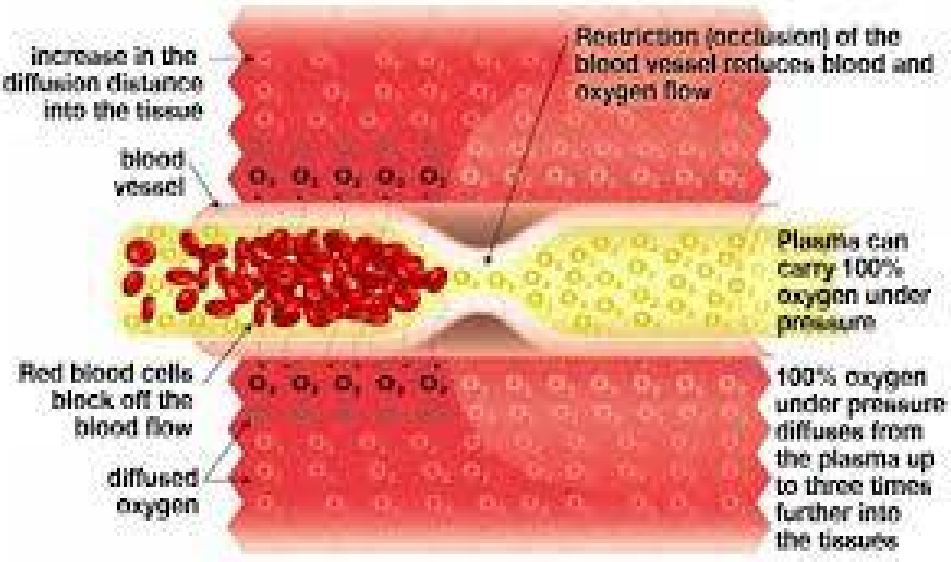



**Fig 10. Hyperbaric oxygen therapy**

Despite these factors the efficacy of HBO for other NSTIs is still controversial .The studies on the efficacy of HBO therapy and its outcome with respect to length of hospital stay and mortality have failed to show statistically significant difference. As a result, HBO is not a mandatory component of therapy, but its use should never delay definitive surgical care.

An HBO treatment regimen usually consists of wound exposure at 2.5 to 3.0 atm and breathing 100% oxygen for 90 mins every 8 hours in the first 24 hours of infection, and then twice daily. HBO is continued for a minimum of 5 days and is discontinued when the patient is stable and has no evidence of going necrosis.

**PROGNOSIS AND OUTCOME**

The mortality associated with NSTIs has been in the range of 16% to 45%. Multiple prognostic factors have been identified, including the presence of clostridial infection. For patients who survive no matter what their premorbid state-major disfigurement and lengthy rehabilitation are common

## CLINICAL FORMS OF NECROTIZING FASCIITIS

### Fourniers gangrene

Fourniers gangrene is an aggressive infection caused by gram negative aerobic bacteria, enterococci, and anaerobic bacteria such as bacteroides spp, and Peptostreptococci. It may be caused by the penetration of GIT or urethral mucosa by bacteria. Extremes of pain may herald the onset of infection which spreads to the anterior abdominal wall and muscles of the gluteal region. In males, infection spreads frequently to the scrotum and penis. Surgical inspection, placement of drains and appropriate surgical debridement are necessary for both diagnosis and treatment. Antibiotic therapy should be based on culture and sensitivity and gram staining when available. An appropriate empirical regimen would be ampicillin or ampicillin and sulbactam combination with metronidazole combination or clindamycin. Alternatively Gram negative coverage is advisable if the patients has been hospitalized in the past or antibiotics have been used recently. This could be alleviated by sustitution of ticarcillin-clavulanic acid or piperacillin-tazobactam for ampicillin or by addition of a fluorinated quinolone or aminoglycoside.

**FIG 10.Fourniers gangrene**

**Meleneys synergestic gangrene**

This is a rarer form of NF occuring in postsurgical state of patients, these leisons are ulcerated , slowly expanding and limited to the superficial fascia, resulting in combination between Staph.aureus and microaerophilic streptococcal synergism. As in other forms of necrotizing infection, the main stay of treatment is antibiotic therapy and surgical debridement.


**Fig 11. Meleneys synergistic gangrene**

**Non Clostridial anaerobic cellulitis**

In this, infection is associated with tissue gas producing aerobic organisms and mixed anaerobes. This foul smelling infection is associated with diabetes mellitus and differs from clostridial cellulitis. Surgical exploration is required to distinguish this condition from necrotizing cellulitis, myonecrosis and necrotizing fasciitis by clostridium species.

**Clostridial cellulitis**

In this, recent surgery or local trauma precedes infection, the most common species causing this entity is Clostridium perfringens. The fascia and the deep muscle s are spared whereas gas is found invariably in the skin. Although clostridial cellulitis is different from clostridial myonecrosis in that there is less systemic toxicity, it is mandatory that

thorough debridement and surgical exploration should be performed to distinguish between these two entities. MRI scan, CT scan and serum creatinine phosphokinase assay may also be useful to determine whether muscle is involved. Treatment is discussed below under gas gangrene.

**Clostridial gas gangrene**

The clostridial soft tissue infection types are

1 .Simple wound contamination or colonization

2. Cellulitis by anaerobic organisms

3. Clostridial gas gangrene

In the first type , simple wound contamination or colonization does not progress to true infection due to many reasons (eg. there may be insufficient devitalized tissue to promote the infection or there may be effective host defense responses or effective medical and surgical management).It occurs very commonly. 30 % to 80% of open wounds has species of clostridial origin.

The second type, anaerobic cellulitis occurs in the presence of sufficient devitalized tissue for growth of clostridial perfringens or other strains. Although infection extends along the fascial planes, bacteremia and healthy tissue invasion does not occur. The necessary for cure and nearly nil mortality is appropriate removal of devitalized tissue at the prompt time.

Myonecrosis or clostridial gas gangrene is the third type. This is defined as an acute invasion of muscle that is healthy and not damaged by previous trauma or ischemia. There are three different subtypes in this

1. Traumatic gas gangrene
2. Spontaneous non traumatic gas gangrene
3. Recurrent gas gangrene caused by clostridium perfringens

Gas gangrene due to trauma is the subtype which is most common 70%. It occurs due to deep penetrating injury compromises blood perfusion which develops an anaerobic environment, which is more favourable for clostridial organisms.

80% of infections are having Clostridium perfringens, and the other remaining patients show the other strains like Clostridium septiscum, Clostridium histolyticum, Closridium novyi, Clostridium faallax, Clostridium bifermentans and Clostridium tertium. The other common predisposing conditions producing traumatic gas gangrene are bowel and biliary surgery, inj adrenaline intramuscular injection, retained placenta, illegal abortion, prolonged rupture of the membranes and intrauterine fetal death or missed abortion in patients who are in the postpartum period.

Spontaneous or non traumatic gas gangrene which is less common is usually caused due to the infection of clostridium septicum aerobic species.

Recurrent gas gangrene due to clostridium perfringens is the type which is least common group.

**Traumatic gas gangrene**

**Pathogenesis**

The trauma initially permits entry of organisms into the deep tissues and develops an aerobic niche which is having a low redox potential and low pH which is acidic for the fast growth of clostridial organisms. The process of necrosis continues within hours .In between the necrotic and normal tissues, few polymorphonuclear leukocytes., yet pavementing of the polymorphonuclear leukocytes along the found endothelium is present within the capillaries and small arterioles and postcapillary venules. Later in the clinical course of illness, leukocytes are also found in the greater vessels. Thus the unique histopathology appearance is clostridial gas gangrene is an early influx of pol morphonuclear leukocytes without adjacent tissue or vascular destruction. This histopathologic picture is different from that seen in pyogenic infections causing infection. Of course later, it is demonstrated that s-toxin at the infection site, destructs the host tissue and the infiltrated inflammatory cells.

This s-toxin favors altered regulations of adhesive interactions between the endothelial cells and polymorphonuclear leucocytes which is present in the circulation or in the nearby tissues and primes white cells for increased activity of respiratory burst.

This in turn produces an injury to the layer of endothelial cells ,stasis of leukocytes with in the vessels and the regional hypoxia of tissues .such decreased perfusion produces an anaerobic environment and in turn causes increased rate of destruction of the marginal tissues. This is unique for clostridial gangrene.

Shock in patients with gas gangrene may be due to the effects of the toxins which are by the theta toxin which is released during the process. The effect is either direct one or an indirect one. Phospholipase C reduces contractility of the heart ex-vivo and may lead to hypotension. WARM SHOCK which is defined as markedly increased cardiac output along with the reduced systemic vascular resistance is produced by the theta toxin

Theta toxin accomplishes this by promoting the endogenous mediators release which causes relaxation of blood vessel smooth muscle such as platelet activating factor or prostacyclin-lipid autocoids. The vascular tone decreases rapidly the compensatory rise in the cardiac output or rapid expansion of the blood volume in the intravascular

compartment occurs in order to maintain tissue perfusion adequate. This kind of adaptive mechanism is seen in gram negative sepsis, however similar kind of compensatory mechanism may not be possible in shock due to C.perfringens as because of toxin alpha directly suppresses on contractility of heart.

**Prevention**

Devitalized wound should be aggressively debridement along with the early repair of any decreased blood flow helps to decrease the gas gangrene in deeply contaminated wounds,

Intramuscular adrenaline prolonged duration of tourniquet application and immediate surgical closure of dirty contaminated traumatic wounds should be avoided.

Patients who are having compound fractures are at increased risk of traumatic gas gangrene , especially if the wound is immediately closed surgically

Patients with contaminated wounds should be treated with adequate and appropriate prophylactic antibiotics

**Clinical findings**

The abrupt onset of severe pain in the recent surgery site or trauma site is the initial symptoms. The mean incubation period is often less than a day but may vary depending upon the degree of the wound

contamination or spillage of bowel contents and the extend of vascular injury with low tissue oxygen tension

First the skin which is pallor changes to bronze than purplish red and becoming tender and tense. Bullae may be clear blue, red or purple.

From the clinical examination and soft tissue imaging gas in the tissue may be obvious. Interestingly none of the MRI, CT has proved to be more specific or sensitive than the clinical finding of crepitus in the soft tissue.

However the imaging study are usually useful for the demonstrating gas in tissues which are present in depth such as the uterus.

Rapid development of features of systemic toxicity occur these includes fever, tachycardia, diaphoresis, shock and multiple organ failure.in 50% of the patients are presented to us with the feature of shock at the time of admission to the hospital. In 15% of the patient bacteremia is seen and can be associated with hemolysis. Not all the cases of C.perfringens gas gangrene are associated with bacteremia. Blood isolate showing C.septicum and C.perfringens where associated with clinically significant wound infections

Hypotension liver necrosis with jaundice and kidney Injury are the further complication of myonecrosis due to clostridial infections. Myoglobinuria and hemoglobinuria develops kidney failure but it may

also be the result of hypotension induced acute tubular necrosis. The toxins can also affect the renal tubular cells directly but this has not been proved

**Diagnosis**

Factors contributing to the diagnosis are increasing pain at the previous Injury are surgery site associated with sign of systemic toxicity and the presence of gas in the tissues.

Demonstrating Gram variable rods at the Injury site is the definite diagnosis although clostridia stain Gram positive when collected from the bacteriologic media when visualized from the tissues which are infected they often stain both Gram Positive and Gram negative. C.perfringens may appear to be encapsulated in fresh collected specimens.

Surgical exploration is inevitable, the exposed muscle looks edematous may be an abnormal reddish blue to black colour and when stimulated does not contract or bleed. Usually some degree of cutaneous and fascial necrosis is also present. Organisms are seen among the degenerating muscle bundles with the absence of acute inflammatory cells in microscopic examination.

## MANAGEMENT

Excellent in vitro activity against C.perfringens and other clostridia have been demonstrated in penicillin, tetracycline, chloramphenicol, metronidazole, clindamycin and numerous cephalosporins.

Randomized trials in human to compare the efficacy of different antibiotics are yet to be conducted. Most literature based on i-vitro data states that penicillin is the drug of choice. However, experimental studied in mice have shown greatest efficacy for clindamycin and least for penicillin.

Erythromycin, rifampicin ,chloramphenicol, tetracycline and metronidazole re agents with greater than penicillin . survival advantage was observed in animals receiving both clindamycin and penicillin , in contrast antagonistic effect was observed with the combination of penicillin and metronidazole. A combination of penicillin and clindamycin is advisable since up to 5% of stains would be resistant to clindamycin. Use of combination of tetracycline and penicillin also advocated based on various experimental studies. Thus, in the absence of clinical trial in humans the best treatment would be administer a combination of clindamycin or tetracycline with the penicillin. Thorough surgical debridement is always mandatory to improve survival, preserve limbs and prevent further complication.

Although some studies have reported better efficacy with HBO therapy when combined with antibiotics and surgical debridement, The use of hyper baric oxygen is controversial. The hyperbaric oxygen therapy of theoretically inhibits bacterial growth, preserve marginally perfused tissue and inhibit toxins productions. Animal studies have demonstrated that HBO alone can be effective treatment If the inoculum is small and treatment is begun immediately on Contrary , other studies have reported that HBO when combine with penicillin was only of slight benefits . However , survival was better with clindamycin lone than with HBO alone , penicillin alone or HBO plus penicillin together.

Strategies in therapy have directed against toxins expression and proadhesive molecules. Target of toxin expression is accomplished in VIVO, by neutralization with specific antitoxins , antibody or by toxin synthesis inhibition. Currently, antitoxins are no longer available. Endogenous pro adhesive molecules may be the promising feature targeted strategy in attenuating toxins induced tissue injury.

**Prognosis**

Gas gangrene of an extremity has a better prognosis than those the truncal or intra-abdominal gas gangrene, because it is hardly feasible to debride such lesions adequately. Truncal gangrene with the associated bacteremia and intravascular hemolysis has the greatest likelihood of

progressing to shock and death.

**Non Traumatic , Spontaneous Gas Gangrene Caused By Clostridium Septicum**

**Pathogenesis**

**Predisposing factors**

- Leukemia
- Cancer chemotherapy
- Colonic carcinoma
- Diverticulitis
- Gastrointestinal surgery
- Lymphoproliferative disorders
- Radiation therapy
- AIDS

Neutropenia associated with spontaneous gas gangrene is due to C.septicum and in such cases commonly found lesions are necrotizing enterocolitis, caecitis and distal ilelitis. These factors allows bacteremia consequently, the aerotolerant C.septicum can establish in healthy tissues.

The important feature of C.septicum different from the toxin of C.perfringens, that alpha toxin of C.septicum has no Phospholipase activity. Active immunization against alpha toxin significantly protects against viable C.septicum experimentally. However he recent cloning and

sequencing of alpha toxin may facilitate further studies in this area,

**Clinical features**

The onset of this disease is often abrupt, with excruciating pain, although the usual symptoms of this patient is heaviness or numbness. Sometimes malaise and confusion may be the first presentation .rapid progression of disease sets in frequently. Haemorrhagic or purplish fluid filled bullae make their appearance. Vascular compromise of the surrounding tissue also causes purple hue of the skin.

**Diagnosis**

Histopathology of muscle and connective tissue reveals the formation of gas and cell lysis, with absence of inflammatory cells.

In spontaneous gangrene, the onset of bacteremia usually occurs before the cutaneous manifestations unlike the traumatic gangrene

Other common conditions presenting with fever and extremity pain in the absence of cutaneous signs of gas gangrene that could be considered are deep venous thrombophlebitis or cellulitis.

**Management**

Human trials evaluating the efficacy of hyperbaric oxygen or various antibiotics for treating spontaneous gas gangrene is yet to be done .The study data indicates that C.septicum are susceptible to penicillin, tetracycline, erythromycin, clindamycin and metronidazole. The aero-

tolerance of C.septicum may reduce the likehood that HBO therapy would be effective.

**Prognosis**

The critical period being the first 24 hours consisting of larger number of death with the mortality ranges from 67% to 100%. Unfavourable factors include conditions underlying compromised immune status and malignancy. Survivors of bacteremia or spontaneous gangrene caused by C.septicum should undergo appropriate diagnostic studies of the gastrointestinal tract. Occasionally this has led to detection and cure of an unsuspected malignancy that might otherwise have been fatal.

**Clostridium sordeii infections**

The characteristic clinical feature of C.sordeii infection are edema and leukemoid reaction, absence of fever, hemoconcentration and even shock in later stages combined with multiorgan failure. Usually C.sordeii infections develop after gynaecological procedures or in postpartum period and mostly represent endometrial infection. Rarely some cases have occurred at minor injured sites such as the laceration of the soft tissues of an extremity. Recently, outbreaks of C.sordeii and C.novyi infections have been found among the intravenous drug users in Scotland, Ireland and England. Patients have presented with soft tissue infections

with shock with a case fatality rate of 20% to 30%. Absence of pain in C.sordeii is typical feature. The absence of fever and the paucity of signs and symptoms make early diagnosis difficult in such infections.

**Clostridium tertium infection**

Clostridium tertium has been associated with spontaneous myonecrosis, however bacteremia in immunocompromised hosts is more common with this infection. Bacteremia probably arises from bowel sources and the presence of the organism in the bowel may be partly related to its relative resistance to penicillin ,clindamycin and cehalosporins. Clostridium tertium usually responds to metronidazole, chloramphenicol and vancomycin. Because this organism can grow in anaerobic environment, it can be by mistake disregarded as a contaminant.

**Pyomyositis**

Most cases of pyomyositis are found in tropical areas. The most common predisposing factor is trauma. First, the seeding of the traumatized muscle occurs and there are no useful clinical finding for specific diagnosis. Within 10 to 20 days, chills, fever, muscle pain and tenderness are manifest. Most patients seek medical care at this stage and a diagnosis can be made by further appropriate imaging studies, needle aspiration or exploration. Patients in whom a diagnosis has not been made

may progress to shock and organ failure. Though these complications are uncommon, Staphylococccus aureus is the most common cause of pyomyositis in tropical and nontropical areas and among HIV positive patients.

Hospitalized immunocompromised patients who are HIV negative occasionally develop pyomyositis caused by gram negative bacteria.

**FIG 13. PYOMYOSITIS OF HIP AND PELVIC MUSCLES**

Surgical drainage of the abscess and empiric administration of parenteral antibiotics such as nafcillin or cephalosporins are reasonable treatments since most cases are caused by Stap.aureus. Definitive treatment can then be established based on cultures and sensitivities. Due to increase in the prevalence of methicillin resistant Stap.aureus, it may be necessary to use vancomycin or linezolid empirically pending sensitivity results.

## MATERIALS AND METHODS

A Prospective Observational study was conducted in Coimbatore Medical College Hospital from July 2016 to July 2017 among the 144 patients admitted to the surgical wards with severe soft tissue infections.

Inclusion Criteria

All patients above 18 years of age with severe soft tissue infections

Exclusion Criteria

1. Patients <=18 years

2. Pregnant patients

Interventions

- Age , sex, site and aetiology of infection, clinical manifestations, comorbidities, predisposing factors, vital signs, laboratory parameters at the time of admission and microbiology of wound and blood cultureshas been recorded

- Culture of pus, aggressive surgical debridement, tissue biopsy, radiological imaging, antibiotic therapy, treatment of complication, amputation or skin grafting were strategized for management.

- The interval between the contact and admission, LRINEC score risk categorization, the time interval between the admission and first surgery, the number of surgical procedures, the need for amputation, the length of hospital stay and the mortality rate had been documented.

- All variables were statistically analyzed further to evaluate the significance of LRINEC score I predicting the clinical outcomes.

# RESULTS

In this study , totally 144 patients presenting to our hospital with soft tissue infections were included in this study. On the basis of Laboratory Risk Indicator for Necrotising Fasciitis (LRINEC), they were evaluated. These patients were classified as Low, Intermediate and High Risk for the onset of Necrotising Fasciitis based on their score. In each category ,patients with infections were managed appropriately and their outcomes are tabulated and discussed as below

- Laboratory Risk Indicator for Necrotising Fasciitis

| Variable | Score |
|---|---|
| C=reactive protein | |
| Positive ≥150 mg/dl | 4 |
| Negative <`150 mg/dl | 0 |
| Total White cell count, | |
| <15 | 0 |
| 15 to 25 | 1 |
| >25 | 2 |
| Hemoglobin,gm/dl | |
| >13.5 | 0 |
| 11 to 13.5 | 1 |
| <11 | 2 |
| Sodium.mmol/l | |
| ≥135 | 0 |
| <135 | 2 |
| Creatinine | |
| ≤1.6 | 0 |
| >1.6 | 2 |
| Glucose,mg/dl | |
| ≤180 | 0 |
| >180 | 1 |

- The score maximum is 13.

- **Based on Score (LRINEC),RISK CATEGORISATION OF PATIENTS**

- The 144 patients in this study were categorised based on the LRINEC stratification as mentioned as below

- **LRINEC SCORE**

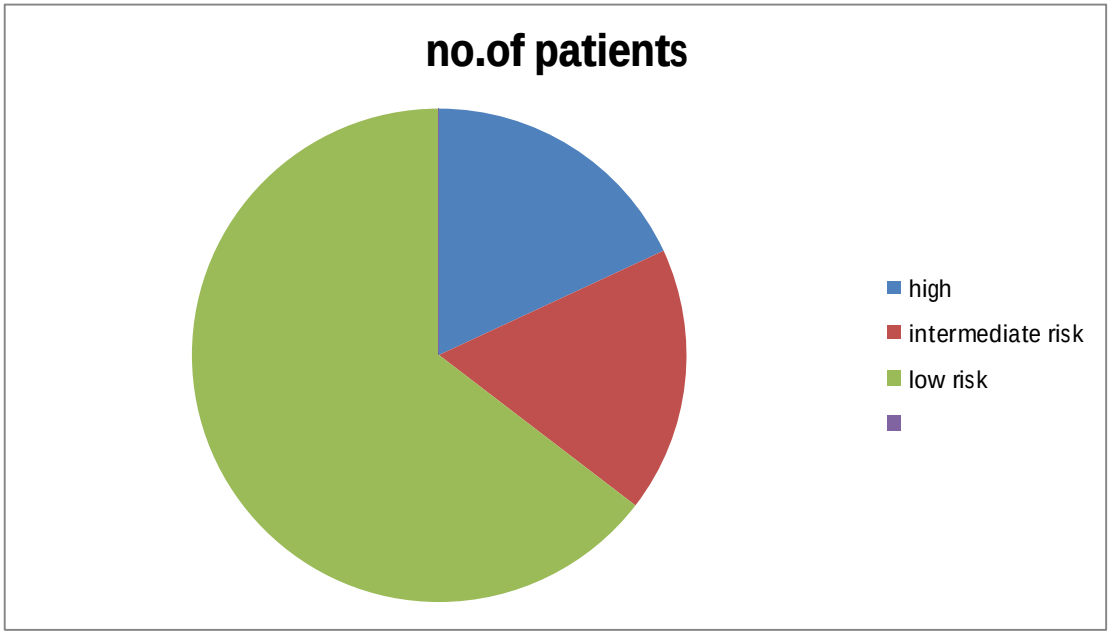
- LOW RISK $\leq 6$

- INTERMEDIATE RISK > 6

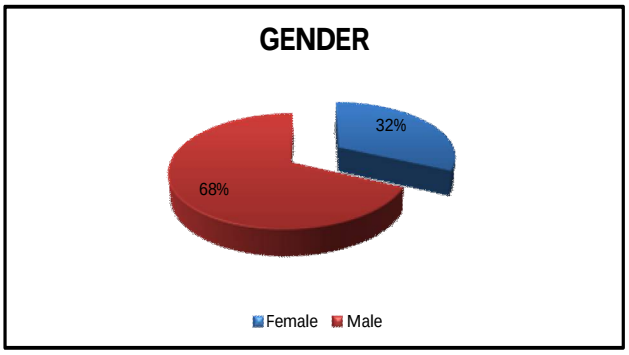
- HIGH RISK > 8

| LRINEC SCORE | NO OF PATIENTS | RISK CATEGORY |
|---|---|---|
| =<6 | 93 | LOW |
| >6 | 26 | INTERMEDIATE |
| >8 | 25 | HIGH |

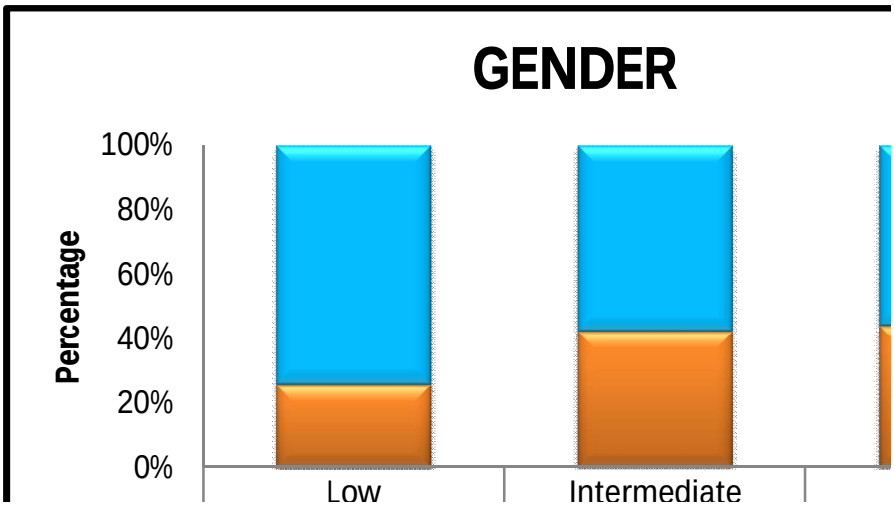
- About 65% of patients with soft tissue infections were categorized as low risk or progression of Necrotizing fasciitis. About 18% and 17% of patients with soft tissue infections were categorized as intermediate and high risk for progression to Necrotizing fasciitis respectively.



## SEX WISE DISTRIBUTION

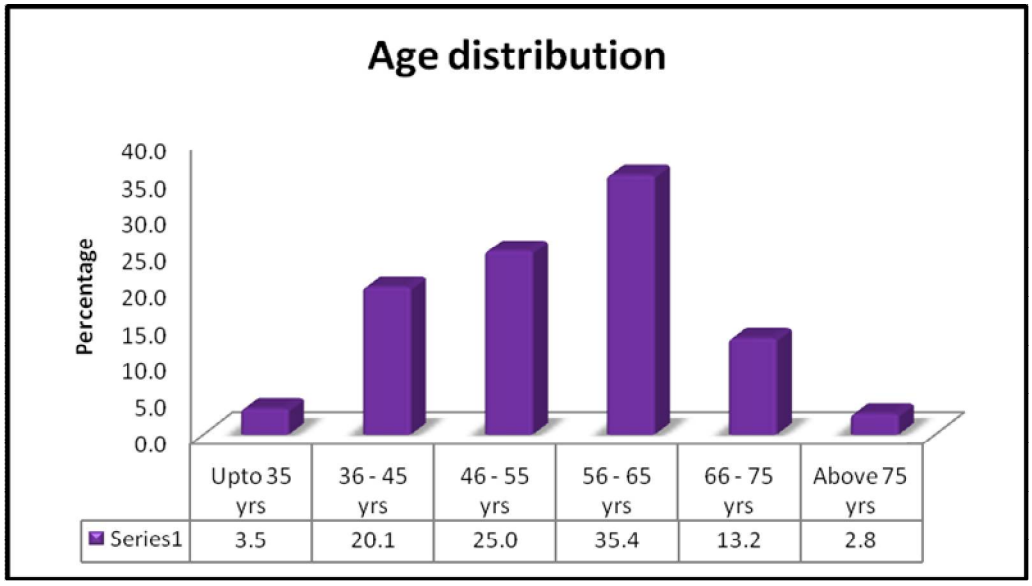



The study population with these soft tissue infections includes 68% males and the rest 32% being females.



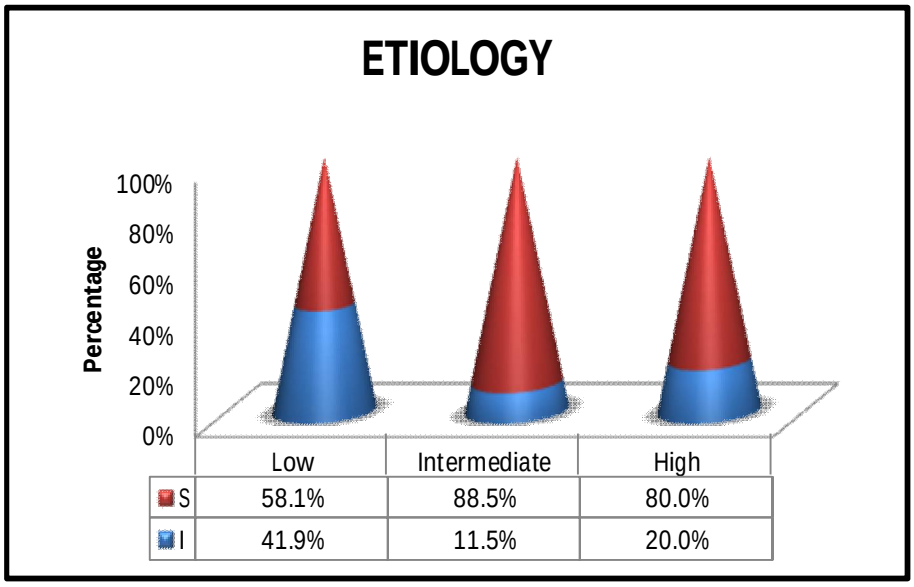

After categorization of study patients into risk groups based on LRINEC score, In low risk, almost 75% are males ant the others being females, and in intermediate and high risk groups, male and female patients have an equal preponderance.

## AGE WISE RISK DISTRIBUTION



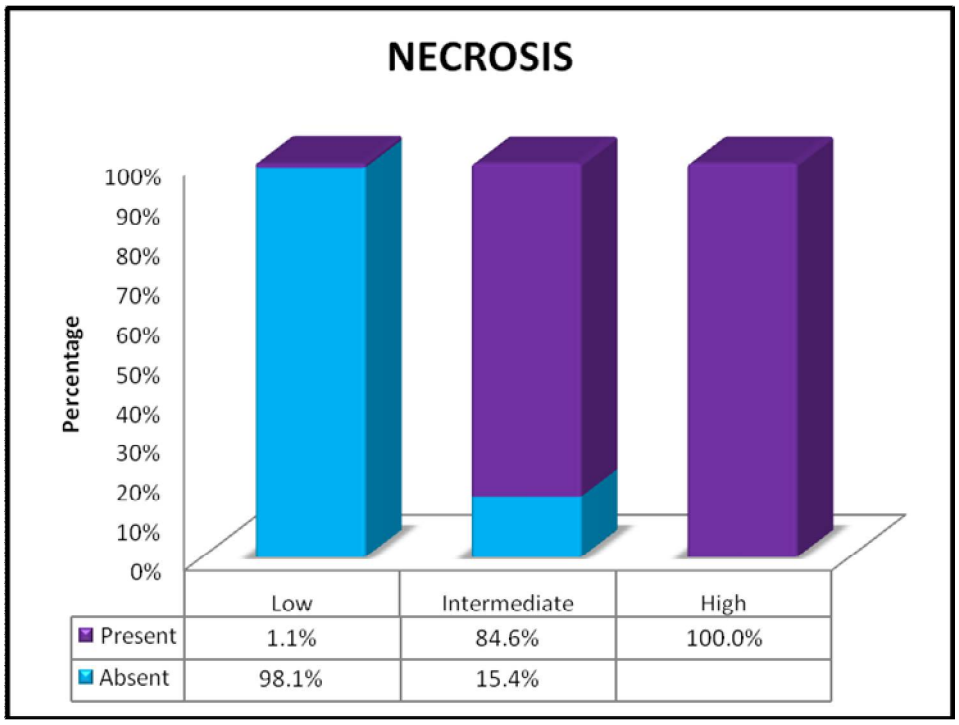

In this study, soft tissue infections occur in all age groups. It appears in the age group between 56 to 65 years who had a high risk of non communicable diseases like diabetes etc. The next common age group involved is 46 to 55 years. It can occur in extremes of age with no age exception. However age beyond 50 years confers the high risk for Necrotizing fasciitis, as evident in this study.

## ETIOLOGY FOR SOFT TISSUE INFECTIONS




Among the patients studied, in low risk, 58.1% of patients had a spontaneous onset of their illness and the other 41.9% had preceding event such as injury more often nail or thorn prick or road traffic accident. In both intermediate and high risk groups, around 80% to 90% of patients had a spontaneous onset. However necrotizing fasciitis is having a spontaneous onset more commonly as proved in this study.

**NECROSIS OF TISSUES IN EACH RISK CATEGORISATION**

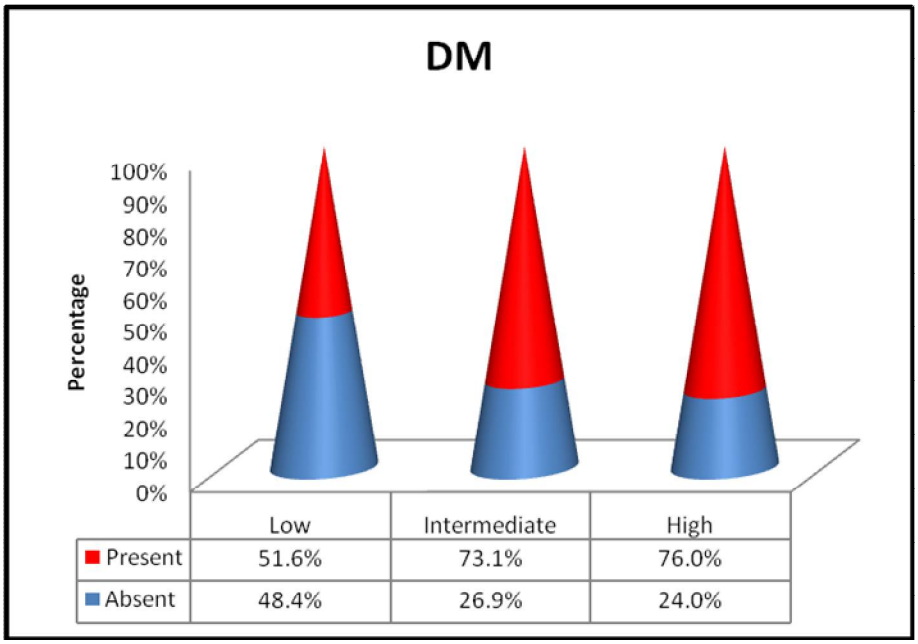



Among the study patients, necrosis of tissues almost absent in low risk patients there is only inflammation. In intermediate groups, about 85% of patients had necrotic tissues and the other 15.4 % had only inflammation but no necrotic tissues. In high risk patients, there was the presence of necrotic tissues in almost 100% of patients.

## CREPITUS IN TISSUES IN EACH RISK GROUPS

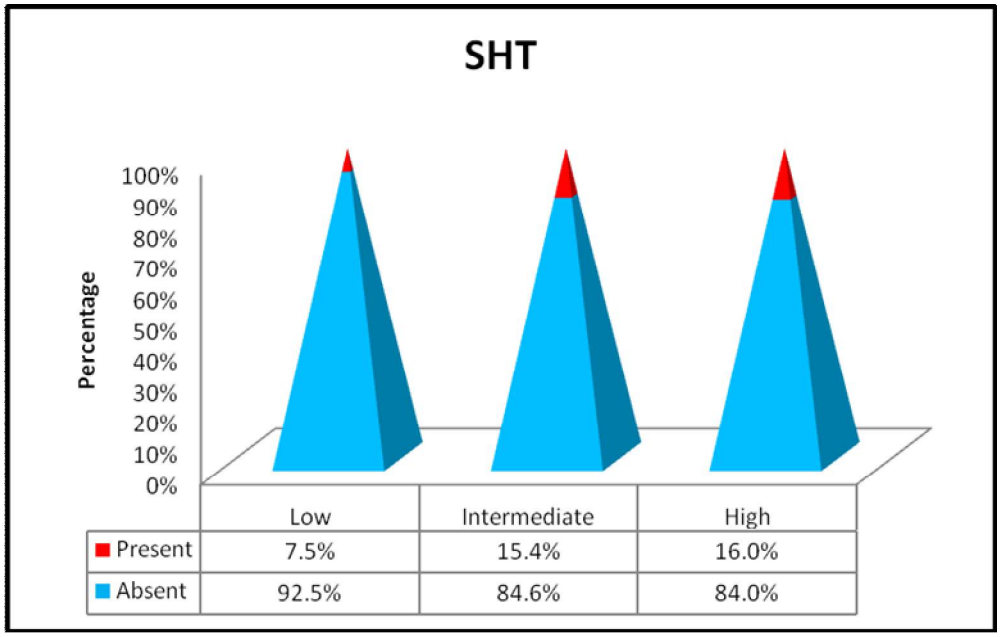
In the study patients , the clinical finding of crepitus is almost absent in both low and intermediate risk groups. In high risk patients around 12% of patients ,crepitus is present in the subcutaneous tissues, suggestive of clostridial infection / gas gangrene. This indicates patients need active intervention in the form of medical and surgical treatment.

## RISK CATEGORISATION IN DIABETIC PATIENT



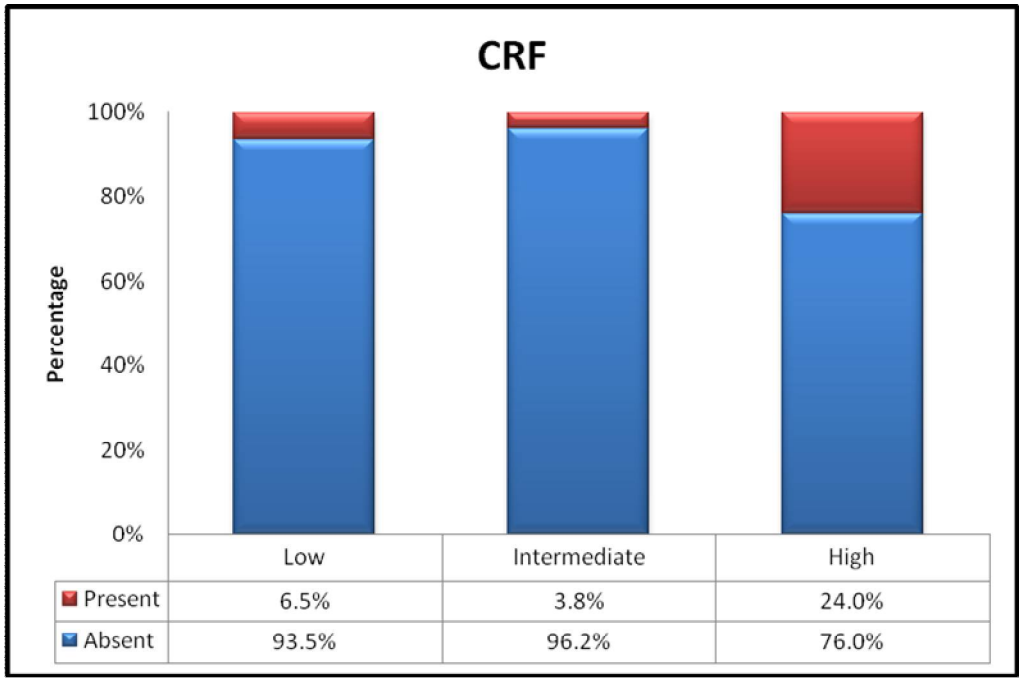

In my study population, around 51% of patients in low risk groups having diabetes, and in both intermediate and high risk almost 75% of patients are having diabetes .By this study we are again proving the presence of diabetes is the important predisposing factor for soft tissue infection and also the progression of infection. High risk groups are found among the diabetic patients.

# SYSTEMIC HYPERTENSION AMONG RISK PATIENTS




In the study group, in low risk groups, 7.5% of patients are having only hypertension. In intermediate and high risk groups around 15% of patients are having hypertension. All others are not having hypertension. This indicates that systemic hypertension alone is not a predisposing factor. When combined with other comorbid conditions, it has some influence in soft tissue infection patients clinical outcomes

# CHRONIC RENAL FAILURE AMONG RISK PATIENTS

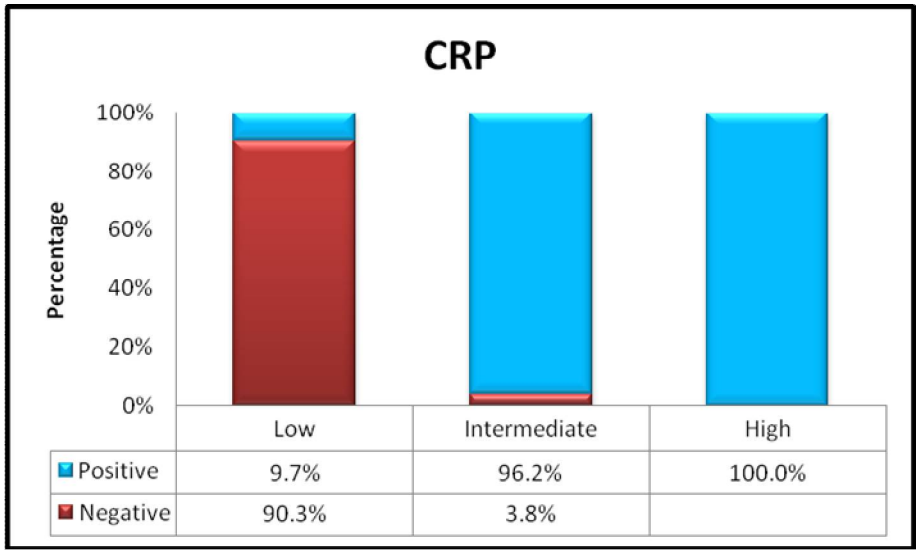



Among the study group, in low and intermediate risk groups, around 4% to 7% of patients having chronic renal failure ,in high risk groups ,chronic renal failure patients are almost 24%. CRF has no much influence in such infections. If coexisted with diabetes ,it is having greater impact in the outcome of disease among the patients.

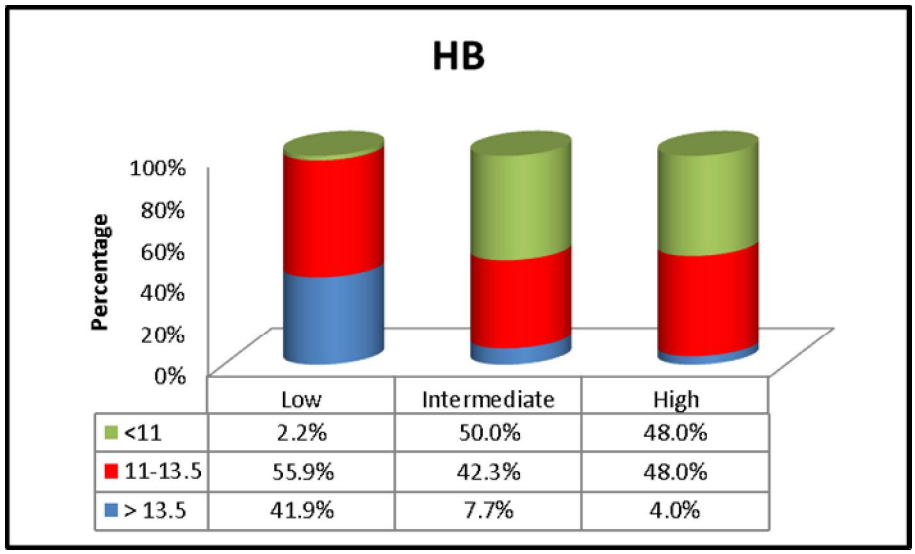
# PERIPHERAL VASCULAR DISEASE AMONG THE RISK PATIENTS

Among the study population ,in all low, intermediate and high risk groups, the incidence of peripheral vascular disease is almost absent. The association between the peripheral vascular disease and the incidence and outcome of soft tissue infections cannot be found.

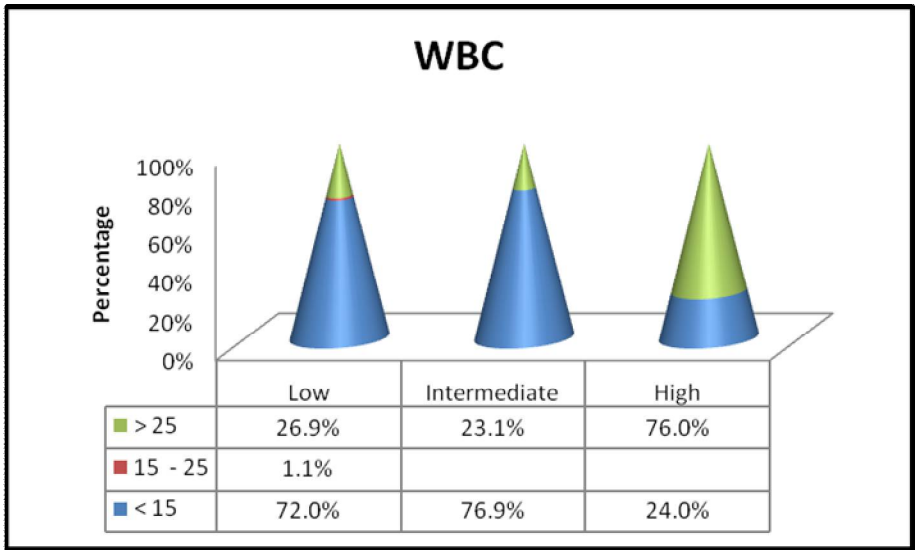
# PARAMETERS OF LRINEC IN RISK CATEGORY




Among these patients ,the CRP will be positive in all high, intermediate and in less than 10% of low risk patients.So CRP is a greater valuable parameter in the score.

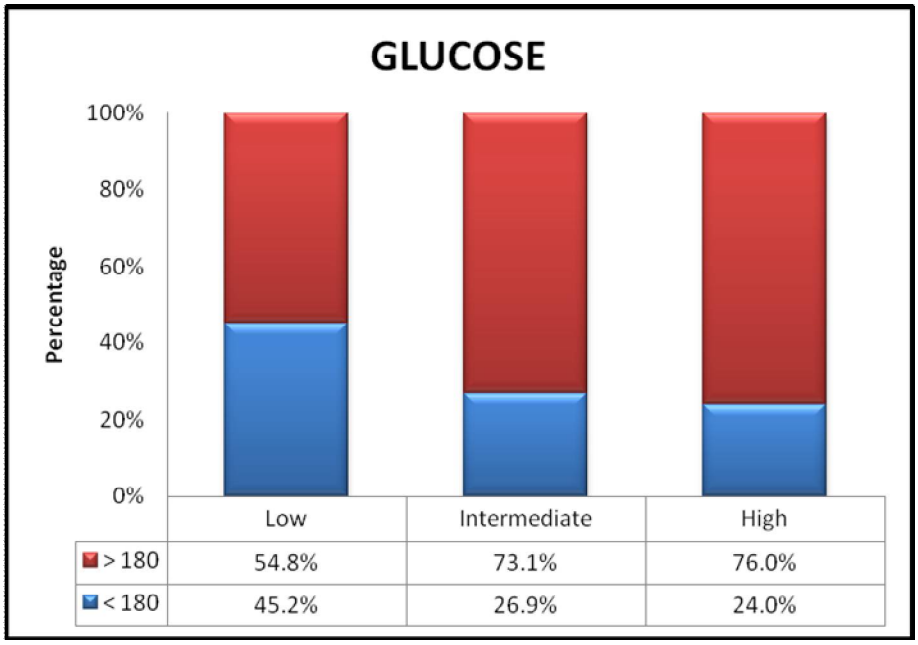


Among the study patients, Hb of <11 gm/dl is present in almost 50% of both intermediate and high risk patients. Almost all the patients in each category had Hb of 11 gm/dl to 13.5 gm/dl. So Hb is considered as the important parameter in this score.

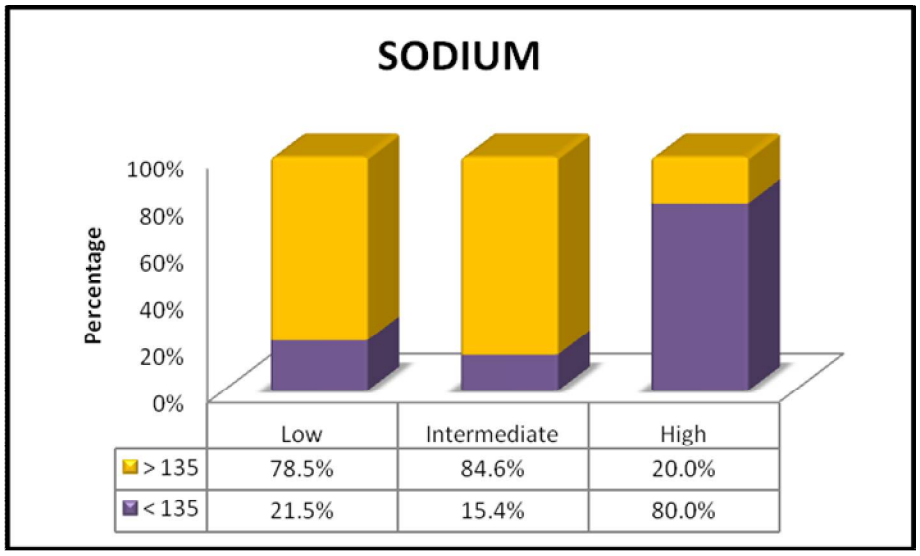



In our study, leucocytosis ( white cell count )is the important parameter in this score. leukocytosis is an early indication of sepsis.

In this study, serum creatinine is one of the component parameter . using chi square test ,serum creatinine had a statistically significant value. Serum creatinine has been taken as a parameter in LRINEC score.

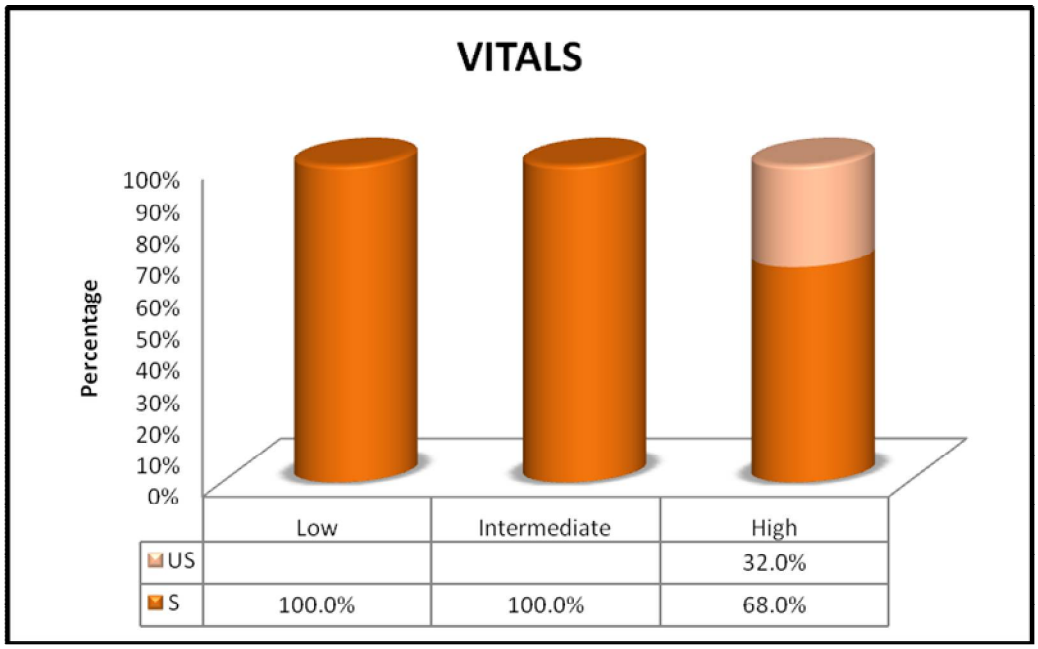



In LRINEC score, patients with soft tissue infections under low intermediate and high risk patients were having 54.8%,73.1%, and 76% of diabetes respectively. So diabetes are an important risk factor for soft tissue infections, so blood glucose levels are the important parameter in LRINEC score and it had a significant p-value using chi-square test.



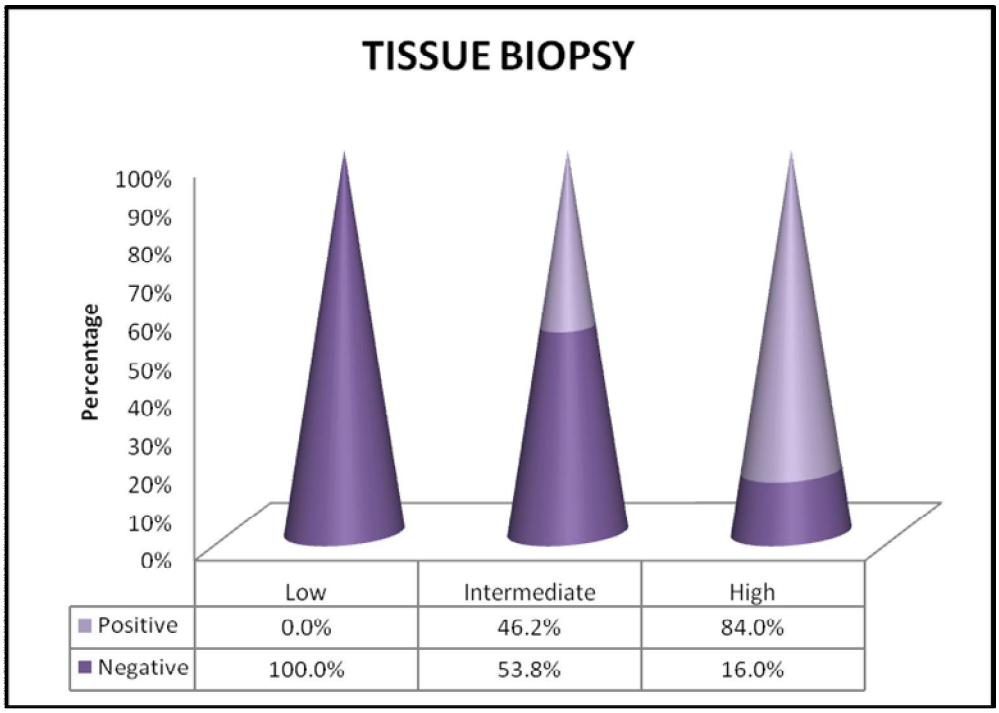

In LRINEC score, serum sodium is one of the important parameter. Using the chi-square test, the calculated value in sodium was found to be statistically significant.So it can be taken as parameter in LRINEC

# VITALS AMONG THE RISK PATIENTS



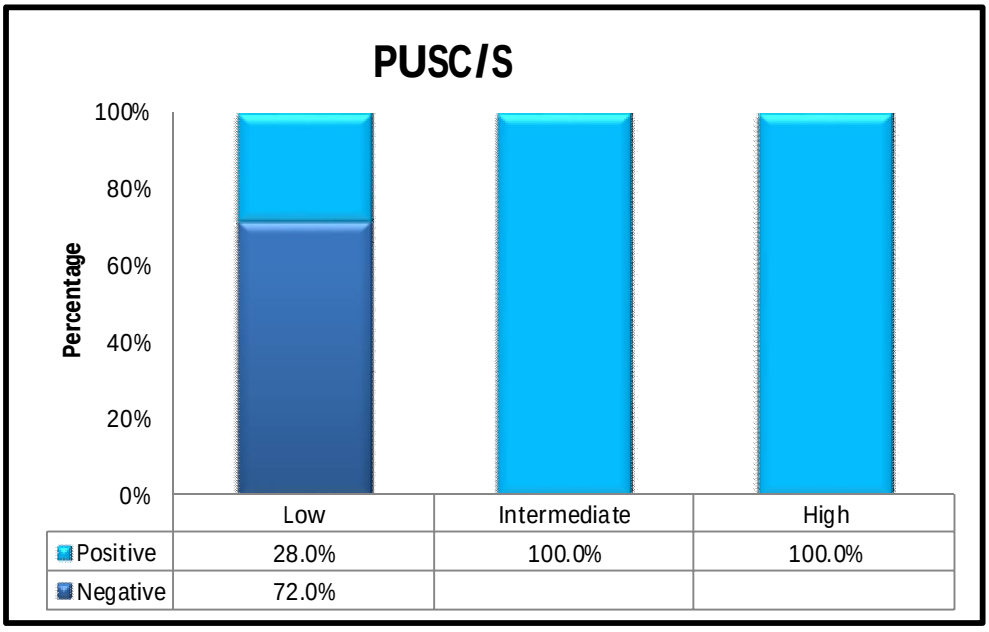

Among the study patients, almost 32% of high risk patients are having unstable vitals at the time of admission. it indicates the need for early resuscititation of patients by giving vasopressors and iv fluids and broad spectrum antibiotics,frequent vitals monitoring are important. Vitals at the time of admission indicates the outcome of high risk patient

# POSITIVE TISSUE DIAGNOSIS IN EACH RISK CATEGORY



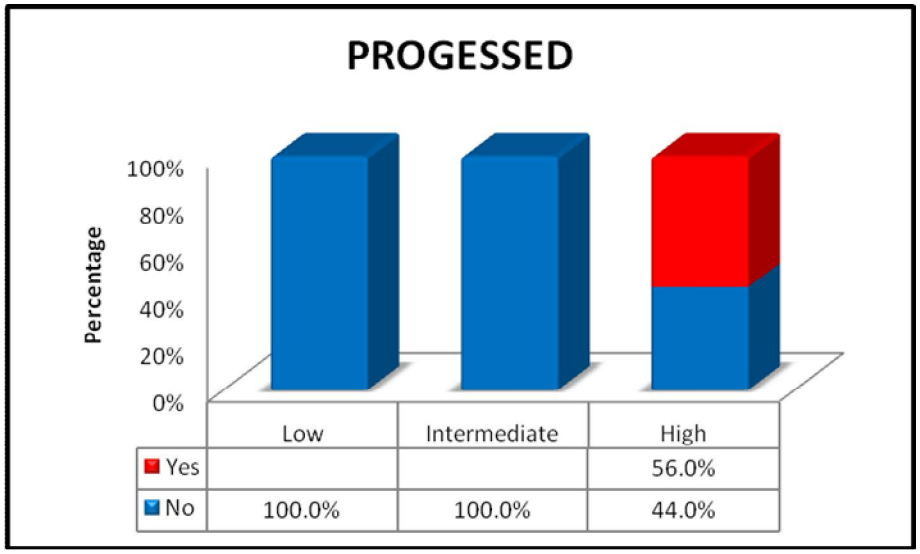

Among the study groups,tissue diagnosis is positive in around 85% of high risk patients based on LRINEC. This score had a highest sensitivity and less than half of patients in intermediate risk groups ,had a positive diagnosis.this indicates that the intermediate group patients had an increased risk of progression to full blown necrotising fasciitis.in low risk groups , none of the patients had positive tissue diagnosis and this signifies that this score had a high specificity.

# PUS CULTURE AND SENSITIVITY IN EACH CATEGORY



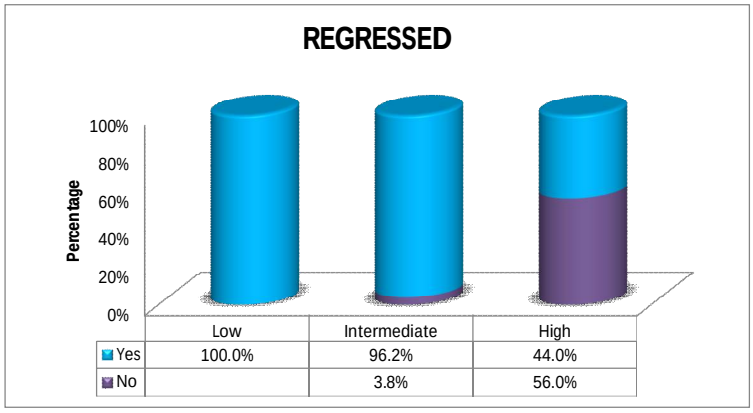

Almost in my study population ,pus c/s had organism growth in both intermediate and high risk patients and in low risk patients, pus c/s are positive for around 30% of patients, and other patients had more features of inflammation, especially in cellulitis.

# OUTCOME – PROGRESSION OF INFECTION AMONG THE HIGH RISK GROUPS




In the study, the low and intermediate groups with medical and surgical debridement was not found to get progressed ,they are improving with our treatment .But almost 56% of patients had progression of disease even with treatment. And these patients had further surgical debridement or inorder to prevent mortality, amputation.

# OUTCOME – REGRESSION OF INFECTION IN THE STUDY PATIENTS



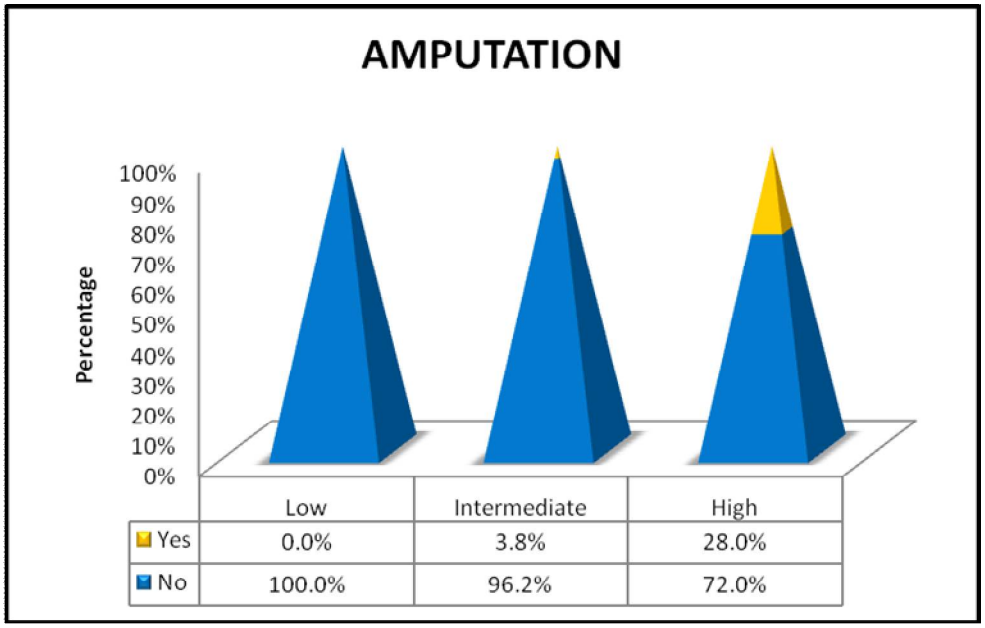

Among the study group ,almost 100% of patients in the low risk group showed the regression of infection. In the intermediate group, after medical and surgical treatment these patients also showed regression to 96.2%

The high risk patients only 44% had the regression of infection after treatment. The other 56% of patients among the high risk patient showed no regression of infection. This emphasises the patient was going in need for removing the septic and necrotic tissue in the form of amputation to prevent mortality

# SSG after debridement in each group

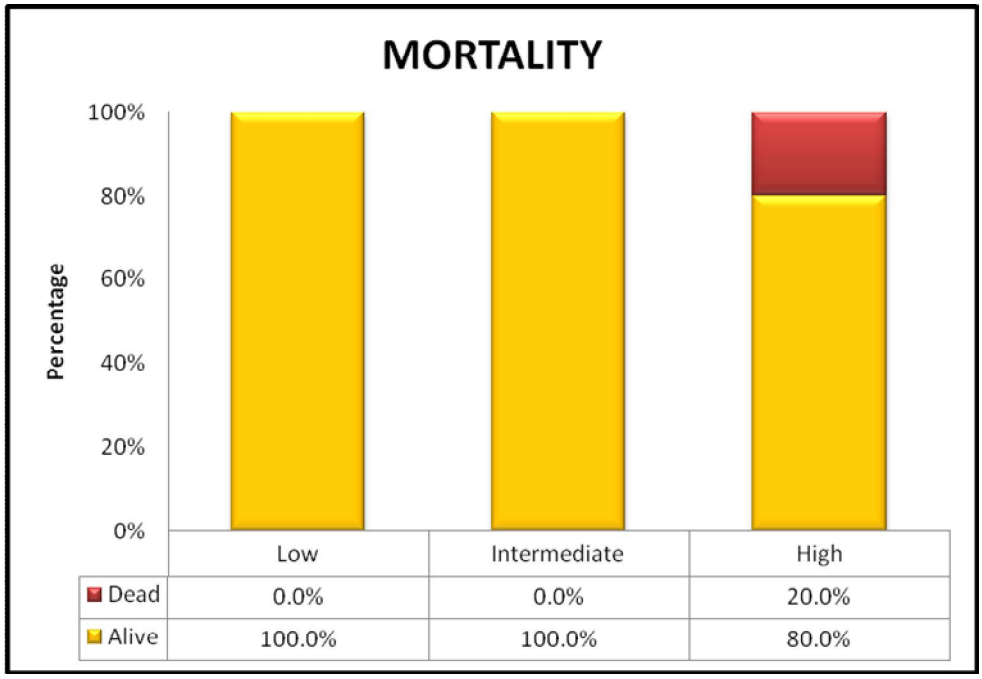
In the study population, the role of SSG in each category after debridement, in low risk category, in most of the cases there was no necrotic tissues, so there was no need for debridement and there was no development of raw area. In low risk group, only 1.1% of patients had undergone SSG. In intermediate and high risk groups ,19.2% and 12% of patients respectively had undergone SSG after debridement.

# AMPUTATION RISK AMONG EACH GROUP



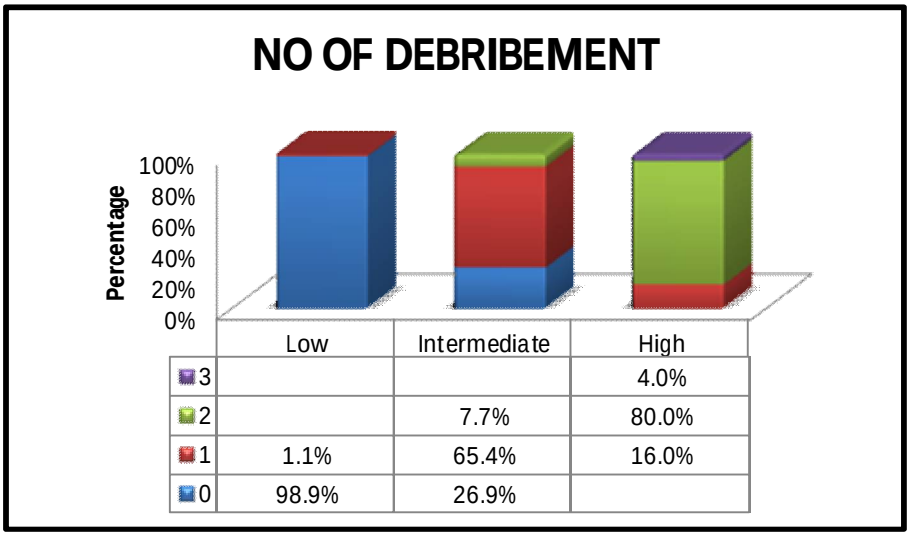

Among the study group, in low risk group, there is no role of amputation as a lifesaving procedure. In intermediate group, almost most of the cases around 97% of patients had a limb salvageable surgeries and only 3% had amputation. In high risk group, inorder to prevent generalised septicaemia and mortality. almost 28% of patients had amputation risk and the remaining 72% of patients had a limb salvageable surgeries.

# MORTALITY AMONG THE RISK CATEGORY




In the study population ,the percentage of mortality is calculated in each risk group .in low and intermediate risk group patients got better with the management and there is no mortality. In high risk group, there is 20% mortality even with treatment and 80% of patients were saved, eventhough they had a serious complications of infection and septicaemia.

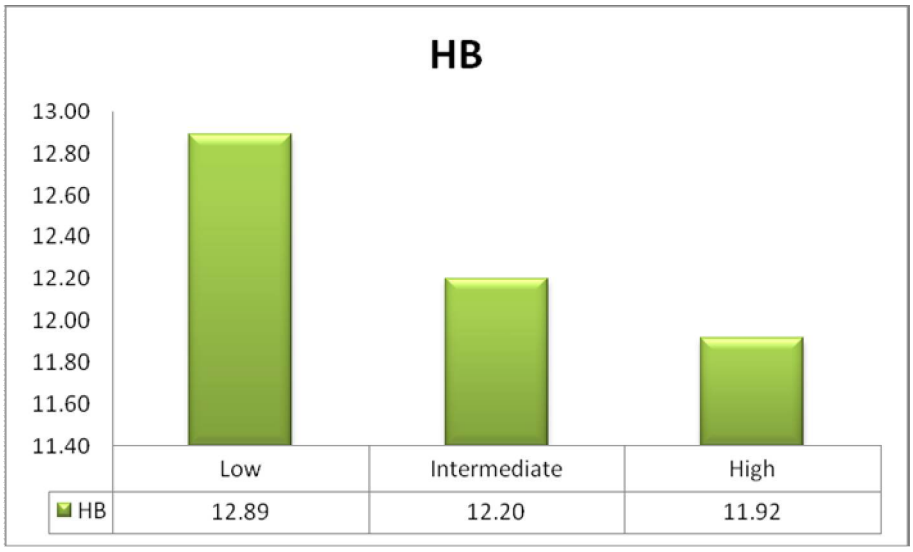
# NO OF DEBRIDEMENT REQUIRED FOR EACH CATEGORY

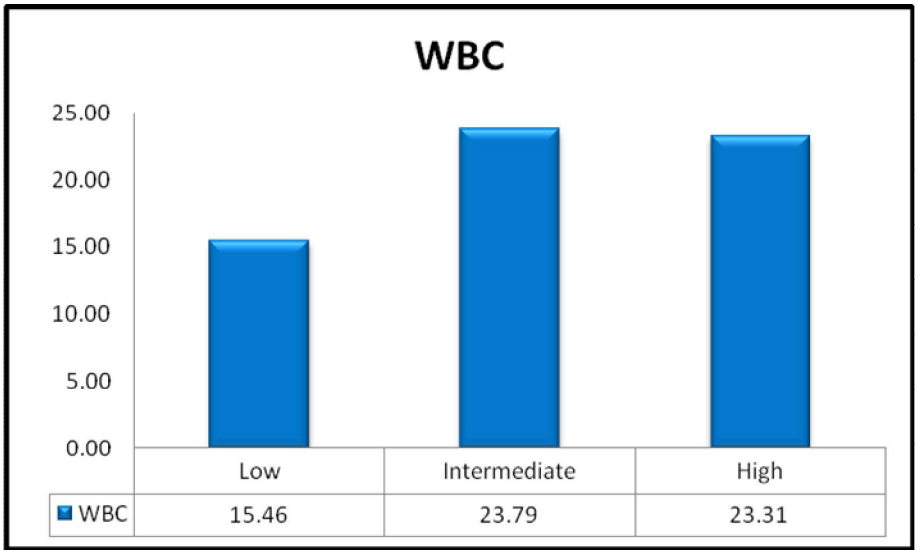


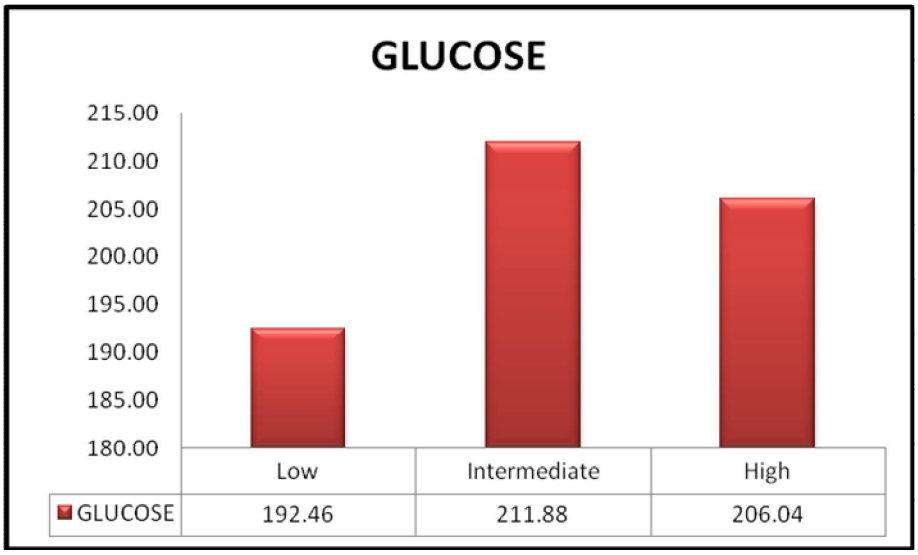

| | Low | Intermediate | High |
|---|---|---|---|
| 3 | | | 4.0% |
| 2 | | 7.7% | 80.0% |
| 1 | 1.1% | 65.4% | 16.0% |
| 0 | 98.9% | 26.9% | |

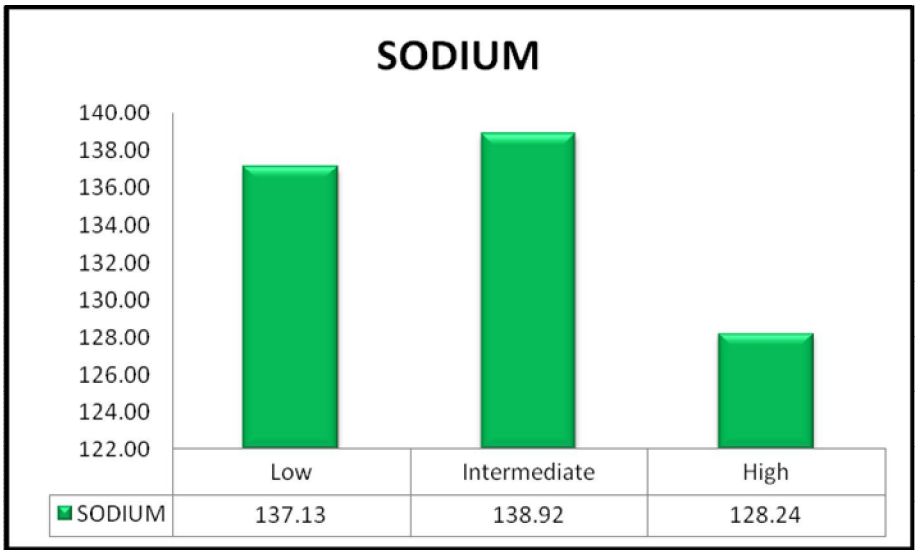
Among the study patients, patients under high risk category group need higher number of debridements than the other risk groups. Almost 98% of patients in low risk category need conservative management, with other 1% of patients requiring few debridement. Patients under intermediate groups had upto 2 debridements to prevent the regression of infection. Patients under low risk category need regular dressing not the extensive debridement only few require It.

# THE MEAN VALUE OF EACH PARAMETERS IN EACH CATEGORY AS SHOWN IN THE BELOW DIAGRAM




GLUCOSE
215.00
210.00
205.00
200.00
195.00
190.00
185.00
180.00
Low
Intermediate
High
GLUCOSE
192.46
211.88
206.04

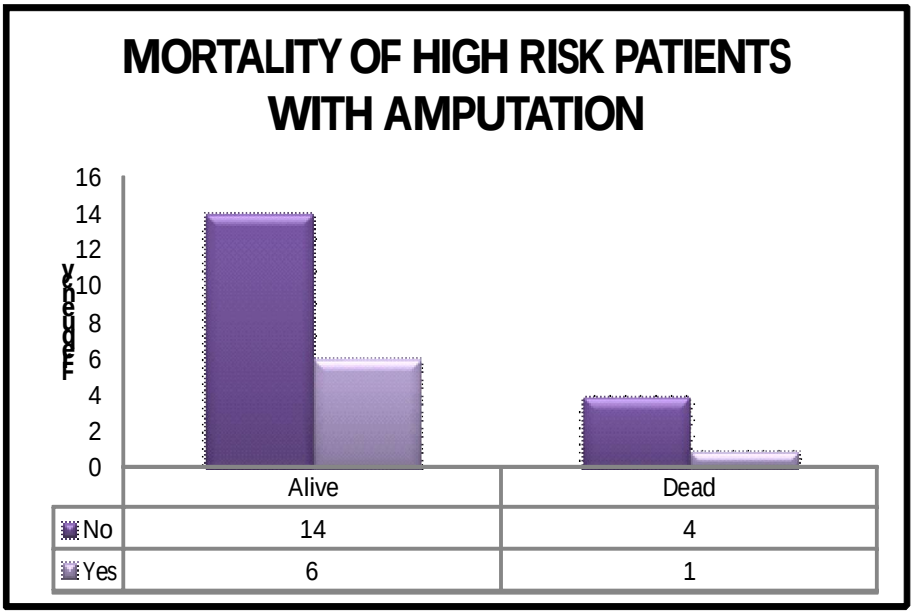

SODIUM
140.00
138.00
136.00
134.00
132.00
130.00
128.00
126.00
124.00
122.00
Low
Intermediate
High
SODIUM
137.13
138.92
128.24

## TAB.4 P-VALE OF THE PARAMETERS OF LRINEC SCORE

# ANOVA

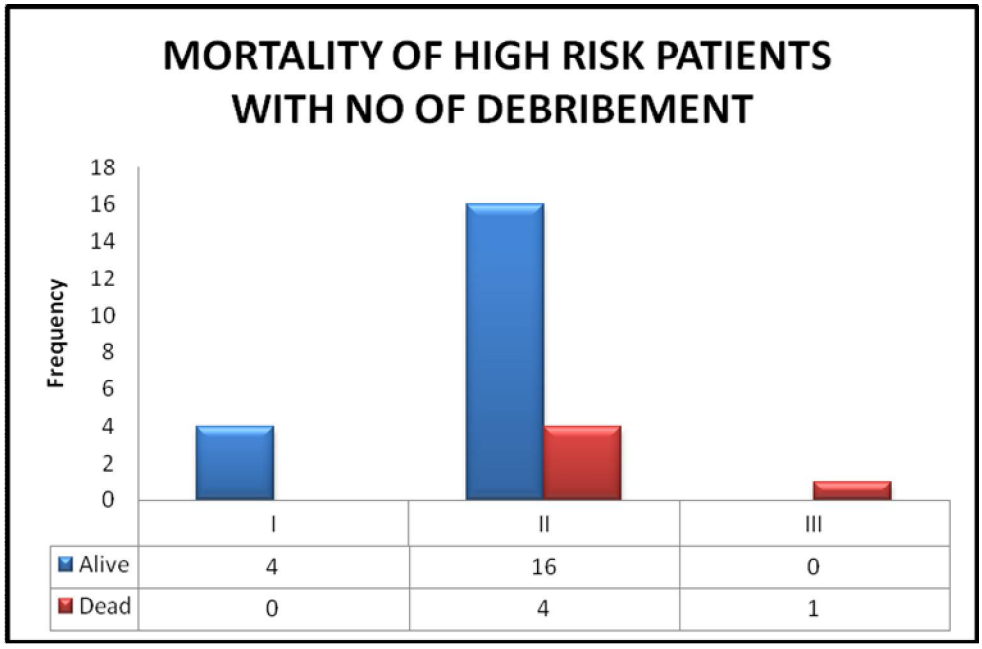
| | | Sum of Squares | df | Mean Square | F | Sig. |
|---|---|---|---|---|---|---|
| HB | Between Groups | 23.576 | 2 | 11.788 | 4.635 | .011 |
| | Within Groups | 358.611 | 141 | 2.543 | | |
| | Total | 382.187 | 143 | | | |
| WBC | Between Groups | 2159.888 | 2 | 1079.944 | 56.883 | .000 |
| | Within Groups | 2676.958 | 141 | 18.986 | | |
| | Total | 4836.846 | 143 | | | |
| CREATININE | Between Groups | 5.459 | 2 | 2.729 | 15.015 | .000 |
| | Within Groups | 25.630 | 141 | .182 | | |
| | Total | 31.089 | 143 | | | |
| GLUCOSE | Between Groups | 9464.928 | 2 | 4732.464 | 2.084 | .128 |
| | Within Groups | 320196.732 | 141 | 2270.899 | | |
| | Total | 329661.660 | 143 | | | |
| SODIUM | Between Groups | 1844.969 | 2 | 922.484 | 18.569 | .000 |
| | Within Groups | 7004.858 | 141 | 49.680 | | |
| | Total | 8849.826 | 143 | | | |
| DURATION OF HOSPITAL STAY | Between Groups | 1578.160 | 2 | 789.080 | 109.097 | .000 |
| | Within Groups | 1019.833 | 141 | 7.233 | | |
| | Total | 2597.993 | 143 | | | |

# MORTALITY AMONG THE HIGH RISK GROUP AFTER AMPUTATION




In the study population , the high risk patients did not show regression of disease even after debridement of necrotic and septic tissues, they developed septicaemia due to persistent septic foci. In order to prevent mortality, we did amputation of the affected part/limb and we saved the patient. In my study, among 25 high patients, 20 patients were saved including 5 patients who have undergone amputation. 5 patients lost their lives, including 1 amputated patient, mortality risk gets decreased after amputation.

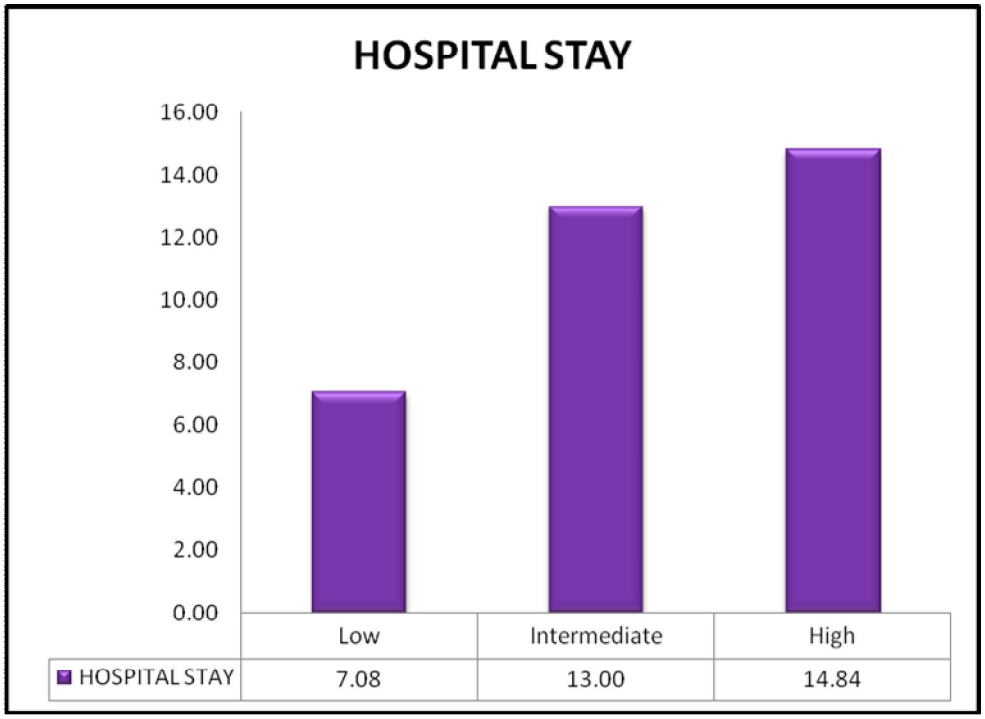
# MORTALITY OF HIGH RISK PATIENTS AFTER DEBRIDEMENT




Among the study population, few high risk patients showed progression of disease after debridement. After two debridement, among the 24 patients, 20 patients were showed the regression, and they were saved, and only 4 patients were dead .after three debridement, one patient was dead. so mortality is more in patients after debridement compared to amputation.

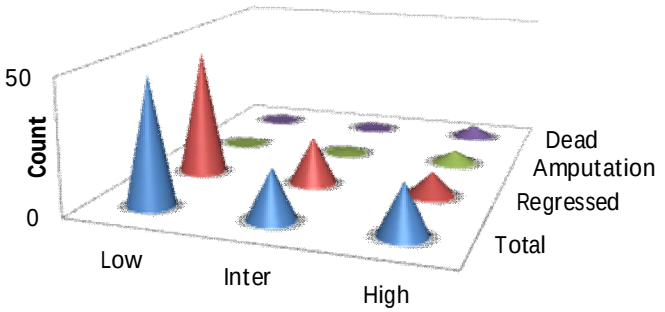
# THE AVERAGE DURATION OF HOSPITAL STAY

# IN EACH CATEGORY




Among the patients, duration of hospital stay is more in high risk patients and intermediate patients the low risk groups. The mean duration of hospital stay is 7 days ,intermediate and high risk groups is 13 to 15 days.

# The Outcome Of  Disease Among Diabetic Patients




| | Low | Inter | High |
|---|---|---|---|
| Total | 48 | 19 | 19 |
| Regressed | 48 | 18 | 9 |
| Amputation | 0 | 1 | 5 |
| Dead | 0 | 0 | 5 |

In this study there are 48  diabetic patients in low risk group, among them, all the patients showed the disease regression  with the medical and surgical treatment.  Among the 19 diabetic patients in the intermediate risk group, 18 patients showed the regression of the disease only one had the progression of the disease and had amputation and no mortality. In high risk group, 19 diabetic patients  , 9 of them showed the regression of the disease and 5 of them  had amputation without mortality. And the remaining  5 of them were dead with rapidly progression of  the disease. Hence diabetic patients were more prone for necrotizing fasciitis and they were having the high mortality rate .

## DISCUSSION

Necrotizing soft tissue infections are fatal progressive infectious processes, most prevalent among diabetic patients, impoverished obese diabetic patients and injection drug users with a varied spectrum of clinical course associated severe sepsis. The associated systemic inflammatory response syndrome in the setting of sepsis causes changes in the biochemical parameters in a predictable manner.

The LRINEC score is a measure of these changes and predicts the presence of necrotizing fasciitis. Other soft tissue infections (eg. cellulitis and abscesses) rarely cause an inflammatory state severe enough to cause such disturbances in the laboratory variables.

This Prospective study of 144 patients with soft tissue infections included ninety eight females (68%0 and forty six females (32%). The mean age group was around 56 to 65 years. Diabetes Mellitus was the most common comorbidity(86 cases).Other comorbid conditions included Chronic Renal Failure (13 cases), Systemic Hypertension (15 cases), Peripheral Vascular Disease (0 cases).The important manifestations at presentation were erythema, edema, tenderness, bullae, necrosis, tachycardia and hypotension. Extremity was the most common site involved followed by scrotum and perineum. About 97 cases (67%) had

soft tissue infections of unknown origin and the remaining 47 cases(33%) were attributed injury as a cause

## TAB.5.The characteristics of the study group under low risk category

| RISK CATEGORY | NO OF CASES | TISSUE DIAGNOSIS | | PUS C/S | | DM | | SHT | | CRF | | ETIOLOGY | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| LOW RISK | 69 MALES & 24 FEMALES | + | 0 | + | 27 | + | 48 | + | 7 | + | 6 | S | 54 |
| | | _ | 93 | _ | 67 | _ | 45 | _ | 86 | _ | 87 | I | 39 |

**Treatment given:**

83 cases were treated conservatively and 10 cases were debrided. no cases were taken for amputation. Most of the cases were treated with intravenous antibiotics. SSG was done for the debrided 10 cases.

Outcome

All cases were improved

**TAB 6.THE CHARACTERISTICS OF THE STUDY UNDER INTERMEDIATE RISK CATEGORY**

| RISK CATEGORY | NO.OF CASES | TISSUE DIAGNOSIS | | PUS C/S | | DM | | SHT | | CRF | | ETIOLOGY | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| INTERMEDI-ATE RISK | 15 MALES & 11 FEMALES | + | 12 | + | 26 | + | 19 | + | 4 | + | 1 | S | 23 |
| | | _ | 14 | _ | 0 | - | 7 | _ | 22 | _ | 25 | I | 3 |

Treatment Given:

19 cases were debrided and 5 cases were treated with SSG. One case was taken for amputation who showed the progression of the disease even after debridement. Rehabilitative measures were started

Outcome

All the cases were improved

**TAB 7. THE CHARACTERISTICS OF THE STUDY UNDER HIGH RISK CATEGORY**

| RISK CATEGORY | NO OF CASES | TISSUE DIAGNOSIS | | PUS C/S | | DM | | SHT | | CRF | | ETIOLOGY | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| HIGH RISK | 14 MALES & 11 FEMALES | + | 21 | + | 25 | + | 19 | + | 4 | + | 6 | S | 20 |
| | | _ | 4 | _ | 0 | _ | 6 | _ | 21 | _ | 19 | I | 5 |

Treatment given:

All cases were debrided and 3 cases were treated with SSG. 7 cases were amputated who had showed the progression of the disease in order to prevent septicaemia and its complications.

Outcome

5 cases were dead and the other cases were saved with debridement or with amputation.

Sensitivity and specificity

LRINEC scoring system has a better sensitivity and positive predictive value in identifying the onset of Necrotizing fasciitis in soft tissue infections Of all the comorbidities, Diabetes Mellitus was the most frequent predisposing factor followed by the Chronic renal failure in both primary and secondary NF in this study group.

**TAB 8. CHARACTERISTICS IN PATIENTS WITH LRINEC SCORE<6 AND PATIENTS WITH >=6**

| Variables | LRINEC<6 | LRINEC>=6 | p-VALUE |
|---|---|---|---|
| MALE SEX | 69 | 29 | 0.102 |
| NECROSIS | 10 | 51 | 0.000 |
| CREPITUS | 0 | 3 | 0.001 |
| VITALS UNSTABLE | 0 | 8 | 0.000 |
| UNKNOWN ETIOLOGY | 54 | 43 | 0.005 |
| DM | 48 | 38 | 0.011 |
| CRF | 6 | 7 | 0.015 |
| SHT | 7 | 8 | 0.308 |
| PVD | 0 | 0 | 0 |
| NO OF DEBRIDEMENT | 0/1/2 | 1/2/3 | 0.00 |
| AMPUTATION | 0 | 8 | 0.000 |
| MORTALITY | 0 | 5 | 0.002 |

- ( p- value can be calculated by using Fischers exact and Pearson chi square methods)

- In the above table , the p-value (0.011) reveals that there is an association between Diabetes and the severity of risk. The

proportion of high/intermediate risk is more in DM group compared to the nondiabetic group. The p-value (0.000) shows that there is an association between the risk of amputation and high risk. The high risk patients are at more for going to the procedure of amputation.

- The p-value (0.000) states there is an relation between the scoring and the risk of mortality. High risk patients had a more chance of going to the complications and death. The cut off of LRINEC >= 6 has better sensitivity and specificity in identifying the risk of the patient.

## Bacteriological studies

- In this study, pus culture and sensitivity was taken before giving antibiotics for all the admitted soft tissue infection patients. The reports were collected after 48 hours or more. The collected data was analysed. The most commonly isolated organisms were Streptococci, E.coli, Staphylococcus, Bacteriodes, Klebsiella pneumonea, Pseudomonas aeruginosa. The most commonly present organism in the diabetic patients is E.coli. According to the sensitivity to the antibiotics , patients were treated.

# CONCLUSION

Necrotizing soft tissue infections are often fatal, characterised by extensive necrosis of the fascia and subcutaneous tissues. It is perhaps the most severe form of soft tissue infection potentially limb and life threatening. Early diagnosis of necrotizing fasciitis is essential to advocate timely management for the better wellbeing of the patient.

LRINEC –Laboratory Risk Indicator for Necrotizing Fasciitis score is based on routine laboratory investigations that are readily available, at most centres that can help distinguish Necrotizing fasciitis from other soft tissue infections.

LRINEC scoring system has a better positive predictive value in identifying the onset of necrotizing fasciitis and risk strategizing of the patients with severe soft tissue infections.

There is statistically significant association between Diabetic Mellitus and the severity of risk.

The significance of LRINEC score in predicting the clinical outcome of the patient can also be outlined in this study and as well as the mortality

Further studies are needed to determine whether additional interventions targeted to the high mortality risk group can lead to improved outcomes.

Finally Laboratory Risk Indicator for Necrotizing fasciitis (LRINEC) score can be used as an adjuvant in the management of soft tissue infections especially in secondary care hospitals and may prevent delayed referral to tertiary centres where experienced surgeons ,infectious diseases and specialists may guide immediate operative and ancillary management ,thereby improving the clinical outcome of the patient.

## APPENDIX-1

## ABBREVIATIONS

| | | |
|---|---|---|
| C. | - | Clostridium |
| CRF | - | Chronic Renal Failure |
| CRP | - | C Reactive Protein |
| CT | - | Computed Tomography |
| DM | - | Diabetes Mellitus |
| ED | - | Emergency Department |
| GIT | - | Gastro Intestinal Tract |
| GAS | - | group A Streptococci |
| HBO | - | Hyperbaric Oxygen |
| IV | - | Intravenous |
| LRINEC | - | Laboratory Risk Indicator For Necrotizing Fasciitis |
| MRI | - | Magnetic Resonance Imaging |
| NF | - | Necrotizing Fasciitis |
| NSTIs | - | Necrotizing soft tissue infection |
| PVD | - | Peripheral Vascular Disease |
| SHT | - | Systemic Hypertension |
| sp. | - | species |
| USG | - | Ultrasonogram |

**Appendix-2**

**PROFORMA**

1) NAME -

2) AGE -

3) SEX -

4)IP NO. -

5) D.O.A -

6)D.O.P -

7) D.O.D -

8) CLINICAL FINDINGS -

a) SITE AND EXTENT -

b) INFLAMMATION -

c)INDURATION -

d) NATURE OF DISCHARGE-

9) VITALS ON ADMISSION

a) TEMP -

b) PULSE RATE -

c) BP -

d) RR -

10) COMORBID FACTORS

a) RECENT H/O INJURY OR SURGERY -

b) SMOKING -

c) ALCOHOL -

d) IF ON STEROIDS -

e) IMMUNOSUPPRESSORS -

f) IV DRUG USE -

11) COMORBID DISEASES

a) DIABETES -

b) MALIGNANCY -

c) CARDIAC DISEASE -

d) PVD -

e) HIV/AIDS -

12) INVESTIGATIONS -

a) TOTAL COUNT -

b) BLOOD SUGAR -

c) HB -

d) SR.CREATININE. -

e) SR.SODIUM -

f) CRP -

g) LRINEC SCORE -

h) PUS C/S -

i) X-RAY LOCAL REGION -

j) HIV/HBSAG STATUS -

13) MANAGEMENT -

a) DEBRIDEMENT -

NO OF DEBIDEMENT -

FREQUENCY AND TOTAL -

b) AMPUTATIONS -

c) SKIN GRAFTING -

14) FINAL OUTCOME

a) STATUS OF THE DISEASED PART -

b) MORTALITY/MORBIDITY -

## APPENDIX-3

## CONSENT FORM

It has been explained to me in my mother tongue and I completely understand my condition, its related complications and the treatment options available. I have been explained in detail regarding this study- **LRINEC – Laboratory Risk Indicator for Necrotizing Fasciitis**- An objective scoring system as a tool for early diagnosis of Necrotizing Fasciitis

I hereby give my consent to participate in the above mentioned study.

Date

Place

Signature of the relative Signature of the patient

With name with name

Signature of the witness with name

# xggpj y;go t k;

vdf;F fPH;f z;l vy;yh fUj;Jf;fS k;vdJ jhapbkhHpahf jkpHp y; vdf;F mwpt[Wj;jggl;lJ. nkYk; ehd; vdJ clyepiyapd;Vwgl;l nehapidg;gwwpak/ mjdhy;Vwgl;Lk; gpd; tpist[fisak;nkYk;nkwpbfhz;L njitggLk;rpfpr;ir Kiwfisg; gwwpak; KGtJkhf mwpe;J bfhz;nld; ,itaidj;jpw;Fk; nkYk; nkwpbfhs;Sk; rpfpr;ir KiwfSf;Fk;KG kdJld;rk;kjk;bjhptpj;Jf;bfhs;fpnwd;

ehs;:

,lk;:

nehahspapd;cwtpdh; nehahspapd;ifbahggk;

ifbaGj;J

## APPENDIX-4

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| S.NO | NAME | AGE/SEX | CLINICAL FINDING | | | COMORBIDITY | | | | | | | LRINE PARAMETERS | | | | | | | | | | | | | | | OUTCOME | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | INFLAMMATION | NECROSIS | CREPITUS | DM | SHT | CRF | PVD | SMOKING | ETIOLOGY | CRP SCORE | CRP | HB SCORE | HB | WBC SCORE | WBC | SCORE FOR CREATININE | CREATININE | SCORE FOR GLUCOSE | GLUCOSE | SCORE FOR SODIUM | SODIUM | TOTAL | RISK | VITALS | SITE OF INFECTION | NO.OF DEBRIBEMENT | TISSUE BIOPSY | PUSC/S | PROGESSED | REGRESSED | SSG | AMPUTATION | MORTALITY | DURATION OF HOSPITAL STAY |
| 1 | KANDHASWAMY | 75/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 2 | 9.1 | 1 | 19.1 | 0 | 1.1 | 1 | 234 | 2 | 119 | 10 | H | S | RT.LEG | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 13 |
| 2 | SATHYANATHAN | 65/M | 1 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 2 | 26 | 2 | 1.8 | 0 | 175 | 2 | 128 | 11 | H | US | LT.LEG | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 12 |
| 3 | JAYA | 72/F | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.6 | 2 | 26 | 2 | 2.6 | 1 | 234 | 2 | 123 | 12 | H | US | PERINEUM | 2 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 8 |
| 4 | VASANTHI | 56/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 4 | POS | 0 | 13.5 | 0 | 7 | 0 | 1.4 | 1 | 184 | 0 | 136 | 5 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 5 | SHANTHI | 42/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 4 | POS | 0 | 14.2 | 0 | 10.1 | 0 | 1.2 | 0 | 134 | 0 | 135 | 4 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 6 | NATHAN | 56/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | I | 0 | NEG | 1 | 12.6 | 0 | 5.6 | 2 | 1.8 | 1 | 296 | 0 | 141 | 4 | L | S | LT UPPERLIMB | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 7 | MURUGAN | 35/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | I | 4 | POS | 1 | 11.6 | 1 | 16.5 | 0 | 1.6 | 1 | 193 | 2 | 131 | 9 | H | S | RT.LEG | 2 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 15 |
| 8 | USHA | 65/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 13.1 | 1 | 18.5 | 0 | 1.5 | 0 | 153 | 0 | 135 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 9 | KUMARAN | 75/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 1 | 22.5 | 0 | 1.3 | 0 | 179 | 0 | 136 | 6 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 10 | JAMUNA | 34/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.6 | 1 | 16.5 | 0 | 1.4 | 0 | 123 | 2 | 123 | 4 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 5 |
| 11 | AJITHKUMAR | 17/M | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.7 | 1 | 18.5 | 0 | 1.1 | 0 | 119 | 2 | 121 | 8 | I | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 16 |
| 12 | KUMAARSWAMY | 60/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 14.1 | 0 | 11.4 | 0 | 0.9 | 1 | 193 | 2 | 133 | 3 | L | S | BOTH LEGS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 13 | SUBRAMANI | 28/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | I | 4 | POS | 2 | 8.4 | 1 | 18.5 | 0 | 0.9 | 1 | 230 | 2 | 131 | 10 | H | S | LT.LEG | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| 14 | SETTU | 45/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 11.7 | 1 | 18.6 | 0 | 0.8 | 1 | 252 | 2 | 129 | 5 | L | S | LT FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 15 | ARUSWAMY | 45/M | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | S | 0 | NEG | 1 | 11.8 | 0 | 12.8 | 0 | 0.7 | 1 | 253 | 0 | 137 | 2 | L | S | RT FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 16 | RANGASWAMY | 50/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.3 | 1 | 16.5 | 0 | 0.6 | 1 | 234 | 0 | 137 | 7 | I | S | RT.FOOT | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 8 |
| 17 | SELVI | 45/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.8 | 0 | 10.8 | 0 | 1.6 | 1 | 312 | 0 | 128 | 2 | L | S | RT FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 18 | LAKSHMI | 35/F | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 2 | 9.1 | 1 | 16.6 | 2 | 1.7 | 1 | 247 | 0 | 137 | 10 | H | S | RT LOWERLIMB | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 15 |
| 19 | KARUUPASWAMY | 60/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 15.6 | 0 | 11.2 | 0 | 1 | 1 | 192 | 0 | 141 | 5 | L | S | BOTH LOWERLIMBS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 20 | SUBBHULAKSHMI | 48/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.8 | 0 | 9 | 0 | 0.8 | 1 | 208 | 0 | 139 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 21 | DAVID | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 4 | POS | 1 | 12.4 | 1 | 15.9 | 0 | 0.6 | 0 | 142 | 0 | 137 | 6 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 22 | BANUMATHI | 57/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 2 | 9.4 | 1 | 16.1 | 0 | 0.7 | 1 | 348 | 0 | 142 | 4 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 23 | NARAYANAN | 42/M | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 1 | I | 4 | POS | 2 | 8.1 | 1 | 18.6 | 0 | 0.9 | 0 | 143 | 0 | 137 | 7 | I | S | LT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 24 | KANNAN | 65/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 2 | 8.4 | 2 | 30.5 | 2 | 1.9 | 0 | 153 | 0 | 140 | 6 | L | S | LT.LEG | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 12 |
| 25 | SENTHILKUMAR | 51/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 13.8 | 0 | 11.5 | 0 | 0.7 | 1 | 194 | 0 | 143 | 5 | L | S | BOTH LEGS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 26 | GANESAN | 46/M | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.1 | 0 | 16.1 | 2 | 1.7 | 1 | 356 | 0 | 139 | 4 | L | S | LT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 27 | VIJAYAKUMAR | 42/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.9 | 0 | 10.7 | 0 | 1.1 | 0 | 165 | 0 | 141 | 1 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 28 | DEVAKUMARI | 52/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 10.8 | 1 | 16.4 | 0 | 1.3 | 1 | 289 | 0 | 139 | 7 | I | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 10 |
| 29 | BALASUBRAMANI | 46/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | S | 0 | NEG | 1 | 11.6 | 1 | 17.1 | 0 | 0.7 | 1 | 245 | 0 | 136 | 3 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 30 | RAVIKUMAR | 60/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 10.6 | 1 | 16.2 | 0 | 0.8 | 1 | 234 | 0 | 138 | 3 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 31 | SUKUMAR | 43/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 11.4 | 0 | 10.5 | 1 | 0.9 | 1 | 187 | 0 | 135 | 3 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 32 | HENRY | 56/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 2 | 10.4 | 1 | 15.1 | 0 | 1.1 | 1 | 204 | 0 | 142 | 4 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 33 | RANI | 43/F | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.8 | 0 | 11.8 | 2 | 1.7 | 0 | 153 | 0 | 137 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 34 | JEYALAKSHMI | 74/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.3 | 0 | 12.3 | 0 | 0.7 | 1 | 236 | 2 | 126 | 4 | L | S | RT.HAND | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 8 |
| 35 | MUTHIAH | 85/M | 1 | 1 | 0 | 1 | 0 | 1 | 0 | 0 | S | 4 | POS | 2 | 10.3 | 1 | 20.5 | 2 | 1.8 | 1 | 183 | 2 | 118 | 12 | H | US | RT.LEG | 2 | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 21 |
| 36 | KUMARAVEL | 56/M | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.7 | 2 | 26.4 | 0 | 0.8 | 1 | 193 | 0 | 142 | 8 | L | S | SCROTUM | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 16 |
| 37 | LAKSHMANAN | 60/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.6 | 1 | 15.9 | 0 | 0.9 | 1 | 230 | 0 | 134 | 2 | I | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 38 | CHINNASWAMY | 65/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 11.3 | 0 | 12.3 | 2 | 1.7 | 0 | 145 | 2 | 119 | 5 | L | S | SCROTUM | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 39 | PALNIAPPAN | 72/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.9 | 0 | 14.8 | 0 | 0.8 | 0 | 173 | 0 | 143 | 1 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 40 | SWAMIAPPAN | 58/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.7 | 0 | 13.4 | 0 | 0.9 | 1 | 203 | 0 | 137 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 41 | VADIVELU | 71/M | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | I | 4 | POS | 1 | 12.1 | 1 | 20.4 | 2 | 2.1 | 1 | 211 | 2 | 127 | 11 | H | S | RT.LEG | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| 42 | TAMILSELVAN | 45/M | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | S | 0 | NEG | 0 | 13.8 | 1 | 16.4 | 2 | 1.7 | 0 | 172 | 2 | 131 | 5 | L | S | LT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 43 | BANARI | 46/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.6 | 1 | 18.4 | 0 | 0.6 | 0 | 163 | 0 | 142 | 1 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 44 | SURESH | 38/M | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.8 | 1 | 16.5 | 2 | 1.9 | 1 | 245 | 0 | 138 | 4 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 45 | DAVID | 49/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 14.7 | 2 | 30.4 | 2 | 1.7 | 1 | 211 | 2 | 121 | 11 | H | US | BOTH LOWERLIMBS | 2 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 12 |
| 46 | ESWARI | 39/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.5 | 1 | 16.3 | 0 | 0.9 | 0 | 174 | 0 | 136 | 2 | L | S | RT.UPPERLIMB | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 47 | NOORJAHAN | 46/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.1 | 1 | 17.4 | 0 | 0.8 | 0 | 180 | 0 | 142 | 2 | L | S | PERINEUM | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 48 | FATHIMA | 56/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 2 | 29.4 | 0 | 0.7 | 1 | 312 | 0 | 137 | 8 | I | S | RT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 14 |
| 49 | MANIKANDAN | 45/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.7 | 1 | 15.3 | 0 | 0.6 | 1 | 191 | 0 | 141 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 50 | BASHEER | 55/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | S | 0 | NEG | 1 | 11.7 | 0 | 11.2 | 0 | 0.9 | 0 | 154 | 0 | 136 | 1 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 51 | PALANIYMMAL | 71/F | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.5 | 2 | 30.5 | 0 | 0.8 | 0 | 146 | 2 | 114 | 9 | H | S | RT.LEG | 1 | 0 | 1 | 1 | 0 | 0 | 0 | 0 | 15 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 52 | BACKIYAM | 80/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.9 | 0 | 14.9 | 2 | 1.7 | 1 | 193 | 0 | 138 | 4 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 53 | KATHIRVEL | 47/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.8 | 0 | 12.3 | 2 | 1.8 | 1 | 188 | 0 | 142 | 3 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 54 | JASMINE | 42/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.9 | 1 | 17.5 | 0 | 0.8 | 0 | 157 | 0 | 145 | 2 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 55 | BOSS | 55/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 13.2 | 1 | 20.7 | 2 | 1.7 | 1 | 261 | 0 | 142 | 9 | H | S | SCROTUM | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 17 |
| 56 | GANAPATHY | 56/M | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | S | 0 | NEG | 0 | 14.1 | 1 | 17.8 | 2 | 2 | 0 | 132 | 0 | 138 | 3 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 57 | MURUGANATHAN | 47/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.7 | 0 | 14.3 | 0 | 1.1 | 1 | 203 | 0 | 146 | 1 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 58 | RENUKAA | 42/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.7 | 1 | 19.7 | 0 | 0.5 | 1 | 198 | 0 | 148 | 7 | I | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 11 |
| 59 | DAISY | 47/F | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | I | 4 | POS | 2 | 10.1 | 1 | 21.9 | 0 | 0.6 | 0 | 146 | 0 | 142 | 7 | I | S | RT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 60 | SELVAM | 56/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 11.7 | 1 | 15.5 | 0 | 0.9 | 1 | 184 | 0 | 142 | 3 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 61 | KRISHNAMOORTHY | 52/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.1 | 1 | 16.9 | 2 | 2.1 | 1 | 285 | 2 | 123 | 7 | I | S | SCROTUM | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 6 |
| 62 | LINGAM | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.8 | 0 | 14.2 | 2 | 1.7 | 0 | 121 | 0 | 136 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 63 | TAMILSELVAN | 37/M | 1 | 1 | 0 | 1 | 1 | 1 | 0 | 0 | S | 4 | POS | 1 | 12.3 | 1 | 18.9 | 2 | 1.9 | 1 | 187 | 0 | 135 | 9 | H | S | RT.LEG | 2 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 17 |
| 64 | MUTHULINGAM | 48/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.7 | 1 | 15.2 | 0 | 1.1 | 1 | 213 | 0 | 138 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 65 | JAIGANESH | 36/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 14.1 | 1 | 17.2 | 2 | 1.8 | 0 | 165 | 2 | 113 | 5 | L | S | RT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 66 | DHARMENDRAN | 40/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.4 | 0 | 14.3 | 2 | 1.7 | 1 | 205 | 2 | 129 | 10 | H | S | RT.LOWERLIMB | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 13 |
| 67 | UDAYAKUMAR | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.9 | 1 | 18.5 | 0 | 0.8 | 0 | 176 | 0 | 139 | 1 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 1 | 0 | 0 | 18 |
| 68 | POONGODI | 61/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.5 | 1 | 15.3 | 0 | 0.9 | 1 | 204 | 0 | 144 | 3 | L | S | PERINEUM | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 69 | BUHARI | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.9 | 0 | 13.9 | 2 | 1.7 | 0 | 172 | 0 | 139 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 70 | CHINNATHAMBI | 45/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | S | 0 | NEG | 0 | 13.7 | 1 | 16.3 | 0 | 1.3 | 1 | 191 | 0 | 142 | 2 | L | S | LT.HAND | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 71 | UMAMAHESWARI | 53/F | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | I | 4 | POS | 1 | 13.2 | 1 | 18.6 | 2 | 1.8 | 0 | 146 | 0 | 135 | 8 | I | S | RT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 14 |
| 72 | GURUNATHAN | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.7 | 1 | 15.4 | 0 | 1.2 | 0 | 120 | 0 | 138 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 73 | LOUIS | 67/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.8 | 1 | 18.6 | 2 | 1.7 | 1 | 182 | 2 | 116 | 6 | L | S | BOTH LEGS | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| 74 | KANMANI | 43/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.7 | 2 | 25.4 | 2 | 1.8 | 1 | 237 | 2 | 132 | 12 | H | US | BOTH LEGS | 2 | 0 | 1 | 1 | 0 | 0 | 1 | 0 | 17 |
| 75 | SANKAR | 72/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 11.8 | 1 | 19.4 | 2 | 1.9 | 1 | 245 | 0 | 146 | 5 | L | S | LT UPPER LIMB | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 76 | NATCHIMUTHU | 73/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.8 | 0 | 12.8 | 2 | 1.8 | 0 | 158 | 0 | 139 | 3 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 77 | RAGAVENDRA | 51/M | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 0 | 13.6 | 0 | 14.3 | 2 | 1.8 | 1 | 203 | 0 | 138 | 7 | I | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 0 | 1 | 0 | 11 |
| 78 | KOOTIAMAL | 69/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.9 | 2 | 29.4 | 2 | 1.8 | 1 | 230 | 2 | 125 | 12 | H | US | LT.LEG | 2 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 15 |
| 79 | BALAMURUGAN | 58/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 14.6 | 1 | 18.4 | 0 | 1.5 | 0 | 174 | 0 | 135 | 1 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 4 |
| 80 | VANAJA | 46/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 14.1 | 0 | 13.7 | 2 | 1.9 | 1 | 203 | 0 | 136 | 3 | L | S | LT.HAND | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 81 | DURAIRAJ | 45/M | 1 | 1 | 0 | 1 | 0 | 1 | 0 | 1 | S | 4 | POS | 0 | 13.6 | 0 | 13.8 | 2 | 1.7 | 1 | 196 | 0 | 138 | 7 | I | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| 82 | MANIKUMAR | 54/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.7 | 1 | 16.4 | 0 | 1.6 | 0 | 128 | 0 | 145 | 2 | L | S | LT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 83 | KAMALAM | 65/F | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | I | 4 | POS | 1 | 12.1 | 1 | 17.3 | 2 | 2.3 | 0 | 132 | 2 | 132 | 10 | H | S | RT.LEG | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 17 |
| 84 | THULASIYAMMAL | 48/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.9 | 1 | 18.5 | 2 | 1.7 | 0 | 134 | 0 | 138 | 3 | L | S | RT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 6 |
| 85 | MARIMUTHU | 61/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | S | 0 | NEG | 0 | 13.7 | 0 | 14.9 | 2 | 1.7 | 1 | 209 | 0 | 142 | 3 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 86 | NANDAKUMAR | 56/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 13.7 | 2 | 30.6 | 2 | 1.8 | 1 | 193 | 0 | 143 | 10 | H | S | SCROTUM | 2 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 15 |
| 87 | ESAKKIAPPAN | 53/M | 1 | 1 | 0 | 0 | 0 | 1 | 0 | 0 | S | 4 | POS | 1 | 13.1 | 1 | 23.4 | 2 | 1.7 | 0 | 172 | 2 | 126 | 10 | H | S | RT.LEG | 2 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 18 |
| 88 | RAMAMOORTHY | 43/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.9 | 0 | 14.8 | 0 | 0.9 | 1 | 187 | 0 | 141 | 1 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 4 |
| 89 | DINAKARAN | 67/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.7 | 0 | 13.8 | 0 | 0.8 | 1 | 184 | 0 | 137 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 90 | RAGHUPATHY | 64/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.8 | 0 | 12.7 | 0 | 0.6 | 0 | 156 | 0 | 138 | 1 | L | S | BOTH LEGS | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 91 | THILAKA | 45/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 13.7 | 2 | 29.7 | 0 | 1.1 | 1 | 238 | 0 | 136 | 7 | I | S | BOTH LEGS | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 10 |
| 92 | JAIKUMAR | 59/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | I | 0 | NEG | 0 | 14.2 | 1 | 15.1 | 0 | 0.7 | 1 | 194 | 0 | 137 | 2 | L | S | RT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 5 |
| 93 | MUTHULAKSHMI | 66/F | 1 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.4 | 0 | 11.8 | 0 | 0.8 | 0 | 162 | 0 | 138 | 1 | L | S | LT.HAND | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 5 |
| 94 | SEETHAAMMAL | 63/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 2 | 10.3 | 1 | 23.8 | 2 | 1.9 | 1 | 211 | 0 | 142 | 10 | H | S | LT.UPPERLIMB | 2 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 13 |
| 95 | YUVARAJ | 55/M | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 1 | I | 0 | NEG | 0 | 13.6 | 1 | 16.8 | 2 | 1.7 | 0 | 182 | 2 | 126 | 5 | L | S | RT.LEG | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 8 |
| 96 | HEMANATH | 50/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.1 | 0 | 13.2 | 0 | 0.7 | 1 | 242 | 0 | 138 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 97 | GOVINDH | 66/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.7 | 1 | 18.6 | 0 | 1.5 | 1 | 238 | 0 | 142 | 7 | I | S | RT.FOOT | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 12 |
| 98 | POOZHUKUZHI | 62/F | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 2 | 10.4 | 1 | 23.7 | 0 | 1.5 | 1 | 264 | 0 | 143 | 8 | I | S | LT.LEG | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 18 |
| 99 | JANAKI | 56/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 13.4 | 0 | 12.3 | 0 | 0.9 | 1 | 297 | 0 | 139 | 2 | L | S | LT.HAND | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 100 | UDHAYAKUMAR | 61/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 13.5 | 0 | 13.9 | 0 | 0.8 | 1 | 211 | 0 | 141 | 2 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 101 | SENTHILKUMAR | 53/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.8 | 1 | 20.9 | 0 | 0.9 | 0 | 167 | 0 | 138 | 6 | L | S | LT.LEG | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 13 |
| 102 | SIVASUBRAMANIYAN | 67/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 1 | S | 0 | NEG | 0 | 13.6 | 0 | 12.7 | 0 | 0.6 | 1 | 235 | 2 | 123 | 3 | L | S | RT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 8 |

| | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 103 | THULASIYAMMAL | 53/F | 1 | 1 | 0 | 1 | 1 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 2 | 30.8 | 0 | 1.1 | 1 | 265 | 0 | 138 | 8 | I | S | LT.LEG | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 15 |
| 104 | RAVI | 47/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 12.1 | 2 | 27.8 | 0 | 1.1 | 1 | 234 | 0 | 146 | 7 | I | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 13 |
| 105 | KANNAN | 58/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 15.8 | 1 | 16.8 | 0 | 0.8 | 1 | 248 | 0 | 137 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 105 | PREMKUMAR | 39/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | S | 0 | NEG | 0 | 14.1 | 1 | 15.6 | 0 | 0.9 | 0 | 128 | 0 | 144 | 1 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 106 | ANITHA | 52/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.4 | 2 | 29.8 | 0 | 1.1 | 1 | 201 | 0 | 141 | 8 | I | S | RT.UPPERLIMB | 2 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 18 |
| 107 | JOHN | 48/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 13.4 | 0 | 13.2 | 0 | 0.9 | 0 | 165 | 0 | 138 | 1 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 108 | BANGARU | 57/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 14.7 | 0 | 14.1 | 0 | 0.8 | 1 | 233 | 0 | 142 | 1 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 109 | BHUVANESWARI | 64/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.2 | 1 | 15.7 | 0 | 0.7 | 0 | 147 | 0 | 137 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 110 | LEELA | 56/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 2 | 28.7 | 0 | 1.2 | 1 | 302 | 2 | 127 | 10 | H | S | RT.LEG | 1 | 1 | 1 | 1 | 0 | 0 | 0 | 0 | 18 |
| 111 | NIVETHA | 43/F | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.9 | 2 | 26.7 | 0 | 1.7 | 0 | 156 | 2 | 123 | 9 | H | S | LT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 15 |
| 112 | SUBRAMANI | 81/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.7 | 2 | 30.7 | 0 | 1.6 | 1 | 287 | 0 | 139 | 8 | I | S | SCROTUM | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 16 |
| 113 | SIVASUBRAMANIYAN | 75/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.7 | 1 | 22.3 | 0 | 1.5 | 1 | 207 | 0 | 142 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 114 | DINESHKUMAR | 49/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 1 | 12.6 | 0 | 13.4 | 0 | 1.4 | 1 | 231 | 0 | 145 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 115 | GOPIKRISHNAN | 52/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.9 | 1 | 15.6 | 0 | 1.2 | 1 | 246 | 0 | 141 | 3 | L | S | RT.FOOT | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 116 | CHINNAMANI | 64/M | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 1 | I | 0 | NEG | 1 | 13.2 | 0 | 14.3 | 0 | 1.1 | 1 | 231 | 0 | 138 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 117 | DEVIKUMAR | 56/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 14.5 | 1 | 16.5 | 0 | 1.5 | 0 | 176 | 0 | 135 | 1 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 119 | NIRMALA | 43/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.2 | 1 | 15.6 | 2 | 1.7 | 0 | 165 | 2 | 124 | 5 | L | S | LT.FOOT | 0 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 8 |
| 120 | VINOTHKUMAR | 46/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 14.2 | 2 | 27.6 | 0 | 1.4 | 1 | 234 | 0 | 138 | 7 | I | S | LT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 12 |
| 121 | BINU | 42/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.6 | 2 | 28.7 | 0 | 1.5 | 1 | 204 | 0 | 137 | 8 | I | S | RT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 14 |
| 122 | PILLAI | 59/M | 1 | 0 | 0 | 1 | 1 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.9 | 1 | 20.6 | 0 | 1.3 | 1 | 211 | 0 | 145 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 123 | UNNIKRISHNAN | 47/M | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | S | 0 | NEG | 0 | 13.8 | 1 | 15.7 | 2 | 1.8 | 0 | 165 | 2 | 123 | 5 | L | S | LT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 124 | NEELAMMAL | 61/F | 1 | 1 | 1 | 1 | 0 | 1 | 0 | 1 | S | 4 | POS | 1 | 13.1 | 2 | 28.7 | 2 | 1.9 | 1 | 250 | 2 | 123 | 12 | H | S | RT.LEG | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 0 | 18 |
| 125 | ARAVIND | 39/M | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 14.1 | 2 | 30.8 | 2 | 2 | 0 | 165 | 0 | 147 | 8 | I | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 12 |
| 126 | SATHISHKUMAR | 49/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.6 | 1 | 17.6 | 0 | 1.1 | 1 | 237 | 0 | 138 | 2 | L | S | RT.FOOT | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 7 |
| 127 | VINODHINI | 53/F | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | S | 0 | NEG | 0 | 13.7 | 1 | 15.7 | 0 | 1.2 | 1 | 274 | 0 | 136 | 2 | L | S | RT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 128 | XAVIER | 72/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 13.1 | 1 | 23.6 | 0 | 1.3 | 0 | 136 | 0 | 136 | 6 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 129 | THIRUMALASWAMY | 83/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | S | 4 | POS | 1 | 11.9 | 2 | 29.7 | 0 | 1.4 | 1 | 195 | 0 | 149 | 8 | I | S | RT.LEG | 1 | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 14 |
| 130 | BHUVANESWARAN | 53/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 15.6 | 0 | 14.3 | 0 | 0.9 | 0 | 145 | 0 | 141 | 1 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 5 |
| 131 | ARULRAJ | 56/M | 1 | 0 | 0 | 0 | 0 | 1 | 0 | 0 | S | 0 | NEG | 1 | 11.9 | 1 | 17.6 | 2 | 1.7 | 0 | 132 | 0 | 144 | 4 | L | S | RT.FOOT | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 132 | THERESA | 57/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.8 | 1 | 15.8 | 0 | 1.8 | 1 | 201 | 0 | 137 | 3 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 8 |
| 133 | CHANDRA | 62/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.6 | 2 | 27.6 | 0 | 1.5 | 0 | 152 | 0 | 135 | 7 | I | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 11 |
| 134 | DHANAKUMAR | 59/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 12.5 | 1 | 17.8 | 2 | 1.7 | 0 | 161 | 0 | 138 | 4 | L | S | LT.HAND | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 9 |
| 135 | MUGUTHAN | 64/M | 1 | 1 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 13.1 | 1 | 23.6 | 2 | 1.8 | 0 | 165 | 0 | 146 | 8 | I | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 17 |
| 136 | MURUGADOSS | 45/M | 1 | 0 | 0 | 1 | 0 | 0 | 0 | 0 | I | 0 | NEG | 0 | 13.8 | 1 | 16.5 | 0 | 0.9 | 1 | 245 | 0 | 147 | 2 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 137 | RAMANI | 68/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 1 | S | 0 | NEG | 1 | 12.9 | 1 | 15.8 | 0 | 1.2 | 0 | 143 | 0 | 145 | 2 | L | S | LT.FOOT | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 7 |
| 138 | SHANMUGAM | 59/M | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | S | 4 | POS | 0 | 13.7 | 2 | 25.7 | 0 | 1.9 | 0 | 134 | 0 | 146 | 6 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 13 |
| 139 | SHANMUGAPRIYA | 45/F | 1 | 0 | 0 | 1 | 0 | 1 | 0 | 0 | S | 0 | NEG | 0 | 14.1 | 1 | 16.5 | 2 | 1.5 | 1 | 186 | 2 | 114 | 6 | L | S | RT.LEG | 0 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 17 |
| 140 | CHITHRA | 64/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 1 | S | 4 | POS | 0 | 13.6 | 2 | 28.5 | 0 | 1.5 | 1 | 203 | 0 | 138 | 7 | I | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 1 | 0 | 0 | 21 |
| 141 | VAIDEKI | 64/F | 1 | 0 | 0 | 0 | 0 | 0 | 0 | 0 | I | 0 | NEG | 1 | 11.9 | 1 | 17.2 | 0 | 1.4 | 0 | 157 | 0 | 139 | 2 | L | S | RT.LEG | 0 | 0 | 0 | 0 | 1 | 0 | 0 | 0 | 6 |
| 142 | RENAGARAJAN | 57/M | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 12.3 | 2 | 26.5 | 2 | 1.9 | 1 | 206 | 2 | 117 | 12 | H | US | SCROTUM | 3 | 1 | 1 | 1 | 0 | 0 | 0 | 1 | 11 |
| 143 | PETCHIMIUTHU | 75/M | 1 | 1 | 1 | 0 | 1 | 1 | 0 | 0 | I | 4 | POS | 1 | 12.4 | 2 | 26.5 | 2 | 2.1 | 0 | 134 | 2 | 123 | 11 | H | US | RT.LEG | 2 | 1 | 1 | 1 | 0 | 0 | 1 | 1 | 18 |
| 144 | VANALAKSHMI | 63/F | 1 | 1 | 0 | 1 | 0 | 0 | 0 | 0 | S | 4 | POS | 1 | 11.9 | 1 | 17.4 | 2 | 2 | 1 | 211 | 0 | 138 | 9 | H | S | LT.LEG | 1 | 0 | 1 | 0 | 1 | 0 | 0 | 0 | 16 |

## APPENDIX- 6

## KEYWORDS TO MASTER CHART

INFLAMMMATION,NECROSIS,CREPITUS,INJURY,SPONTANEOUS,DM,SHT,PVD,SMOKING

0 - ABSENT

1 - PRESENT

VITALS

S – STABLE

US –UNSTABLE

C-REACTIVE PROTEIN

POS (POSITIVE) -SERUM LEVEL>=150 MG/DL

NEG (NEGATIVE)- SERUM LEVEL <150 MG/DL

RISK CATEGORY

L –LOW RISK

I – INTERMEDIATE RISK

H –HIGH RISK

TISSUE BIOPSY

0 - NEGATIVE

1 – POSITIVE FOR NF

PUS C &S

0–GROWTH ABSENT

1–GROWTH PRESENT

OUTCOME

PROGRESSED

0 –NO PROGRESSION

1 – PROGRESSION

REGRESSION

0 –NO REGRESSION

1 – REGRESSION

SKIN GRAFT

0 –NOT DONE

1 – APPLIED

AMPUTATION

0 –NOT DONE

1 – AMPUTATION DONE

MORTALITY

0 – ABSENT

1 – PRESENT

DM –DIABETES MELLITUS

CRF – CHRONIC RENAL FAILURE

SHT – SYSTEMIC HYPERTENSION

PVD – PERIPHERAL VASCULAR DISEASE

RBS - RANDOM BLOOD SUGAR

HB –HAEMOGLOBIN

CRP – C-REACTIVE PROTEIN

NF – NECROTIZING FASCITIS

DURATION OF HOSPITAL STAY IN DAYS